

1 UNITED STATES BANKRUPTCY COURT
1 FOR THE WESTERN DISTRICT OF NORTH CAROLINA
2 CHARLOTTE DIVISION
2

3
4 IN RE:)
4)
5 GARLOCK SEALING TECHNOLOGIES)
5 LLC, et al,) No. 10-BK-31607
6)
6 Debtors.) VOLUME II-B AFTERNOON SESSION
7 _____)
7
8

8 TRANSCRIPT OF ESTIMATION TRIAL
9 BEFORE THE HONORABLE GEORGE R. HODGES
9 UNITED STATES BANKRUPTCY JUDGE
10 JULY 23, 2013
10
11
11

12 APPEARANCES:

14 On Behalf of Debtors:

15 GARLAND S. CASSADA, ESQ.
15 Robinson Bradshaw & Hinson, PA
14 16 101 North Tryon Street, Suite 1900
16 Charlotte, North Carolina 28246
15

17 JONATHAN C. KRISKO, ESQ.
16 18 Robinson Bradshaw & Hinson PA
18 101 North Tryon Street, Suite 1900
17 19 Charlotte, North Carolina 28246
19

18 20 LOUIS ADAM BLEDSOE, III, ESQ.
20 Robinson Bradshaw & Hinson PA
19 21 101 North Tryon Street, Suite 1900
21 Charlotte, North Carolina 28246
20

22 RICHARD C. WOLF, ESQ.
21 23 Robinson Bradshaw & Hinson, PA
22 23 101 North Tryon Street, Suite 1900
23 24 Charlotte, North Carolina 28246
24
25

1 APPEARANCES (Continued):
2 On Behalf of the Debtors:
3 RAY HARRIS, ESQ.
3 Schachter Harris, LLP
3 400 East Las Colinas Blvd.
4 Irving, Texas 75039
4
5 CARY SCHACHTER, ESQ.
5 Schachter Harris, LLP
6 400 East Las Colinas Blvd.
6 Irving, Texas 75039
7
7 C. RICHARD RAYBURN, JR., ESQ.
8 Rayburn Cooper & Durham, PA
8 227 West Trade Street, Suite 1200
9 Charlotte, North Carolina 28202
9
10 SHELLEY KOON ABEL, ESQ.
10 Rayburn Cooper & Durham, PA
11 227 West Trade Street, Suite 1200
11 Charlotte, North Carolina 28202
12
12 ALBERT F. DURHAM, ESQ.
13 Rayburn Cooper & Durham, PA
13 227 West Trade Street, Suite 1200
14 Charlotte, North Carolina 28202
14
15 ROSS ROBERT FULTON, ESQ.
15 Rayburn Cooper & Durham, PA
16 227 West Trade Street, Suite 1200
16 Charlotte, North Carolina 28202
17
17 JOHN R. MILLER, JR., ESQ.
18 Rayburn Cooper & Durham, PA
18 227 West Trade Street, Suite 1200
19 Charlotte, North Carolina 28202
19
20 ASHLEY K. NEAL, ESQ.
20 Rayburn Cooper & Durham, PA
21 227 West Trade Street, Suite 1200
21 Charlotte, North Carolina 28202
22
22 WILLIAM SAMUEL SMOAK, JR., ESQ.
23 Rayburn Cooper & Durham, PA
23 227 West Trade Street, Suite 1200
24 Charlotte, North Carolina 28202
24
25

1 APPEARANCES (Continued.):

2 On Behalf of Interested Parties:

3 Carson Protwall LP:

3

4 JULIE BARKER PAPE, ESQ.

4 Womble Carlyle Sandridge & Rice, PLLC

5 P.O. Drawer 84

5 Winston-Salem, North Carolina 27102

6

6 Coltec Industries Inc.:

7

7 DANIEL GRAY CLODFELTER, ESQ.

8 Moore & Van Allen, PLLC

8 100 North Tryon Street, Suite 4700

9 Charlotte, North Carolina 28202-4003

9

10 HILLARY B. CRABTREE, ESQ.

10 Moore & Van Allen, PLLC

11 100 North Tryon Street, Suite 4700

11 Charlotte, North Carolina 28202-4003

12

12 MARK A. NEBRIG, ESQ.

13 Moore & Van Allen, PLLC

13 100 North Tryon Street, Suite 4700

14 Charlotte, North Carolina 28202-4003

14

15 EDWARD TAYLOR STUKES, ESQ.

15 Moore & Van Allen, PLLC

16 100 North Tryon Street, Suite 4700

16 Charlotte, North Carolina 28202-4003

17

17 Creditor Committees:

18

18 Official Committee of Asbestos Personal Injury Claimants:

19

19 LESLIE M. KELLEHER, ESQ.

20 Caplin & Drysdale, Chartered

20 One Thomas Circle NW, Suite 1100

21 Washington, DC 20005

21

22 JEANNA RICKARDS KOSKI, ESQ.

22 Caplin & Drysdale, Chartered

23 One Thomas Circle NW, Suite 1100

23 Washington, DC 20005

24

24

25

1 1 APPEARANCES (Continued.):
2 2 Official Committee of Asbestos Personal Injury Claimaints:
3 3 JEFFREY A. LIESEMER, ESQ.
3 3 Caplin & Drysdale, Chartered
3 4 One Thomas Circle NW, Suite 1100
4 4 Washington, DC 20005
4 5
5 5 KEVIN C. MACLAY, ESQ.
5 6 Caplin & Drysdale, Chartered
6 6 One Thomas Circle NW, Suite 1100
6 7 Washington, DC 20005
7 7
7 8 TODD E. PHILLIPS, ESQ.
8 8 Caplin & Drysdale, Chartered
8 9 One Thomas Circle NW, Suite 1100
9 9 Washington, DC 20005
9 10
10 10 TREVOR W. SWETT, ESQ.
10 11 Caplin & Drysdale, Chartered
11 11 One Thomas Circle NW, Suite 1100
11 12 Washington, DC 20005
12 12
12 13 JAMES P. WEHNER, ESQ.
13 13 Caplin & Drysdale, Chartered
13 14 One Thomas Circle NW, Suite 1100
14 14 Washington, DC 20005
14 15
15 15 ELIHU INSELBUCH, ESQ.
15 16 Caplin & Drysdale, Chartered
16 16 600 Lexington Avenue, 21st Floor
16 17 New York, New York 10022
17 17
17 18 NATHAN D. FINCH, ESQ.
18 18 Motley Rice, LLC
18 19 1000 Potomac Street, NW, Suite 150
19 19 Washington, DC 20007
19 20
20 20 GLENN C. THOMPSON, ESQ.
20 21 Hamilton Stephens Steele & Martin
21 21 201 South College Street, Suite 2020
21 22 Charlotte, North Carolina 28244-2020
22 22
22 23 TRAVIS W. MOON, ESQ.
23 23 Moon Wright & Houston, PLLC
23 24 227 West Trade Street, Suite 1800
24 24 Charlotte, North Carolina 28202
24 25
25 25

1 APPEARANCES (Continued.):

2 Official Committee of Asbestos Personal Injury Claimaints:

3 RICHARD S. WRIGHT, ESQ.
3 Moon Wright & Houston, PLLC
4 226 West Trade Street, Suite 1800
4 Charlotte, North Carolina 28202

5 ANDREW T. HOUSTON, ESQ.
6 Moon Wright & Houston, PLLC
6 227 West Trade Street, Suite 1800
7 Charlotte, North Carolina 28202

8 SCOTT L. FROST, ESQ.
8 Waters Kraus, LLP
9 222 North Sepulveda Boulevard, Suite 1900
9 El Segundo, California 90245

10 JONATHAN A. GEORGE, ESQ.
11 Waters Kraus, LLP
11 3219 McKinney Avenue
12 Dallas, Texas 75204

13 Future Asbestos Claimaints:

14 KATHLEEN A. ORR, ESQ.
14 Orrick, Herrington & Sutcliffe, LLP
15 1152 15th Street, N.W., Columbia Center
15 Washington, DC 20005-1706

16 JONATHAN P. GUY, ESQ.
17 Orrick, Herrington & Sutcliffe, LLP
17 1152 15th Street, N.W., Columbia Center
18 Washington, DC 20005-1706

19 Official Committee of Unsecured Creditors:

20 DEBORAH L. FLETCHER, ESQ.
20 FSB Fisher Broyles, LLP
21 6000 Fairview Road, Suite 1200
21 Charlotte, North Carolina 28210

22

23

24

25

I N D E X

1	1		
2	2	DEBTORS' WITNESSES	PAGE
2	3	THOMAS SPORN	
3	3	Direct Examination By Mr. Schachter	404
3	4	Cross Examination By Mr. Finch	441
	4	Cross Examination By Mr. Guy	496
4	5	Redirect Examination By Mr. Schachter	497
	5		
5	6	LARRY RAY LIUKONEN	
6	6	Direct Examination By Mr. Harris	500
7	7		
8	8		
9	9		
10	10		
11	11		
12	12		
13	13		
14	14		
15	15		
16	16		
17	17		
18	18		
19	19		
20	20		
21	21		
22	22		
23	23		
24	24		
25	25		

1 TUESDAY AFTERNOON, JULY 23, 2013

2 (Court called to order at 2:15 p.m.)

3 THE COURT: Start with the next witness.

4 MR. SCHACHTER: May I proceed, Your Honor?

5 THE COURT: Yes, sir.

6 MR. FINCH: Your Honor, one housekeeping matter
7 before we begin. Defendants -- or Garlock has not been
8 providing us with power points that it's going to use on its
9 experts. I'm fine with that as long as the sauce rule is in
10 effect and we don't have to give our power points. We asked
11 about that last week and there was no agreement about that so
12 I am perfectly fine to proceed on that basis.

13 MR. CASSADA: That's fine. I thought we did have an
14 agreement on it, but that's absolutely fine.

15 THE COURT: Well, if you can work out one, great.
16 If not we'll just do it the same, yes.

17 MR. SCHACHTER: Your Honor, we call Dr. Thomas Sporn
18 to the stand.

19 THE COURT: Okay.

20 THOMAS SPORN,
21 being first duly sworn, was examined and testified as follows:

22 DIRECT EXAMINATION

23 BY MR. SCHACHTER:

24 Q. Good afternoon, Dr. Sporn. Would you please introduce
25 yourself to the court.

1 A. Good afternoon, Mr. Schachter. My name is Dr. Thomas
2 Sporn.

3 Q. And where do you practice medicine?

4 A. I practice medicine at Duke University Medical Center up
5 the road in Durham, North Carolina.

6 Q. What is your position there, sir?

7 A. I am a pathologist. I'm an associate professor of
8 pathology within the medical school and I'm an attending
9 pathologist at the hospital medical center.

10 Q. On the slide we have here it says you're the head of the
11 section of pulmonary and thoracic pathology at Duke
12 University. What are your activities in that capacity?

13 A. Well, I'm a physician who's involved in direct patient
14 care. I don't have my own patients. I don't -- people don't
15 come to see me in clinic. But I'm in charge of -- the lead
16 pathologist in issuing diagnoses at patients who come to Duke
17 Hospital who have thoracic disease.

18 That can mean that I interpret small biopsy specimens. I
19 can interpret small cytology specimens, a few cells that have
20 come off the patient's chest wall into their airway, and as
21 part of an evaluation for lung cancers. I examine whole
22 organs. I examine patients who have died in our hospital with
23 suspected thoracic disease. So I'm a physician who is in
24 charge with the diagnostic end of taking care of folks with
25 diseases of the chest.

1 Q. Are you board certified in any specialties?

2 A. I'm board certified in internal medicine. I'm also board
3 certified in anatomic pathology and forensic pathology.

4 Q. How long have you been at Duke, sir?

5 A. Since 1993.

6 Q. Does this briefly summarize your education? If you can
7 talk us through it in very brief terms. We're trying to be
8 efficient.

9 A. Sure. In brief terms I never for the life of me thought
10 I would end up as a pathologist. Very few people who go to
11 medical school do. I started out my training with the
12 expectation that I was going to be a treating doctor. And
13 I've always had an interest in thoracic disease and trauma and
14 critical care, and that's how I began my initial training
15 after graduating from Georgetown School of Medicine.

16 I stayed on as a resident in internal medicine. I
17 completed the fellowship in chest disease and critical care
18 medicine.

19 And following a brief period out in private practice, I
20 decided the science of medicine was more in line with my skill
21 set and I returned to academics, to Duke where I studied
22 pathology.

23 I was appointed medical examiner for the County of Durham
24 here in North Carolina and later as one of the assistant state
25 chief medical examiners down the road in Chapel Hill.

1 I returned to Duke to complete the remainder of my
2 training, including a fellowship or period of advanced
3 training in lung pathology under the auspices of Dr. Victor
4 Roggli, and I've remained there ever since.

5 Q. Sir, there was a time when you mentioned that you were a
6 treating physician for a brief period of time in private
7 practice. When was that?

8 A. That was in 1992.

9 Q. Where was that?

10 A. That was out on the West Coast in Puget Sound in
11 Bremerton, Washington.

12 Q. Did you have an occasion ever to treat people that had
13 mesothelioma?

14 A. I did. The major employer in the town of Bremerton is
15 the Puget Sound Naval Shipyard. At this point in our
16 country's career we're not building so many battleships and
17 aircraft carriers anymore as we are decommissioning and
18 mothballing them. And a great many of the men and women who
19 worked in the shipyard were decommissioning our old warships
20 from the second world war and beyond, and a lot of them had
21 asbestos-associated diseases. So in my patient population I
22 had asbestotics, I had patients with pleural fibrosis, I had
23 patients with mesothelioma.

24 Q. Did that have a role in your decision to pursue further
25 education and training?

1 A. Yes. As a city kid from Washington, DC, I hadn't had
2 much experience in that regard, and it became quite
3 interesting to me.

4 For the brief time that I was out in Washington state I
5 struck up a friendship with a very famous pulmonary
6 pathologist out there who has a special interest in asbestos
7 disease, Dr. Sam Hammar, and it was Sam that convinced me that
8 pulmonary pathology would be a very attractive career goal for
9 myself and he urged that I consider looking at Duke, amongst
10 other places.

11 Q. And at Duke have you had a specialty in your research
12 interests?

13 A. Well, I'm not a researcher in terms that I run a lab and
14 work with animal models and gels and things like that. My
15 research has been observational and reporting on diagnostic
16 techniques and our experience in our own little division with
17 asbestos disease and mesothelioma.

18 Q. Being at Duke, do you have an opportunity to encounter
19 mesothelioma on a regular basis?

20 A. Yes, sir, very much so. I mean -- and that's due to the
21 interest of the -- some of the treating oncologists and
22 thoracic surgeons at my hospital. We see a tremendous amount
23 of referral cases from the shipyard areas, both in South
24 Carolina and Southern Virginia, as well as elsewhere in the
25 country.

1 So I see -- I see a tremendous amount of mesotheliomas.
2 I was working on one yesterday as I was preparing for this
3 trial in terms of diagnosis and I was also examining a lung
4 yesterday of a gentleman who had had a radical extrapleural
5 pneumonectomy. So I see a tremendous amount of mesotheliomas
6 just in my own hospital and on a consultative and referral
7 basis from pathologists who might not be as familiar with the
8 disease and send me cases to look at for diagnostic purposes.

9 Q. And over the years, have you also participated in
10 consulting for litigation purposes and received specimens of
11 tissue for that?

12 A. Yes I have.

13 Q. And what percentage of the consultation historically is
14 related to plaintiff's work as opposed to defense work from
15 the asbestos litigation?

16 A. Well, it's interesting. You know, neither Dr. Roggli nor
17 myself who handled the lion's share of the consultative
18 practice in that regard at our hospital really sort of bill
19 ourselves as being experts specifically for plaintiffs or
20 experts for defense counsel. We answer the phone. People
21 send us cases. We try to do the best job that we can.

22 When I first began cutting my teeth, so to speak, as a
23 fellow under Victor Roggli, the vast majority of our cases
24 were from plaintiffs. I can't think of in my early going how
25 many cases, if any, we looked at for defense.

1 And in the late '80s -- excuse me, the late '90s, most of
2 the contention was based on the diagnosis. Defense counsel
3 did not want to believe that the plaintiff, in fact, had
4 mesothelioma. And in those days, the reports that we wrote --
5 and we wrote reports for all the, what I call the major
6 players in the asbestos litigation field. We wrote reports
7 for plaintiffs for Peter Angelos. We wrote them for Waters
8 and Kraus. We wrote them for Baron and Budd. We wrote them
9 for Evan Yegelwel and some other Florida based attorneys.

10 Almost all the work I did early on was for plaintiffs
11 explaining to them that we were able to issue a secure
12 diagnosis of mesothelioma for their clients and, if possible,
13 to go the extra mile and prove that it was related to
14 asbestos.

15 Q. And how could you as a pathologist prove it was related
16 to asbestos from pathology?

17 A. There are histologic cues that you can look at. And as
18 pathologists, you don't work in a vacuum. You don't just look
19 at the tissue itself. You examine the entirety of the medical
20 record just as you would if you were dealing with your own
21 patient. And that would include looking at radiographs. That
22 would include looking at occupational exposure history. And
23 then that would also look for things such as plaque or
24 asbestos bodies or increased asbestos fiber burden analysis.

25 Q. Now, you mentioned something that we're going to talk a

1 lot about, asbestos fiber burden analysis. Please explain to
2 the court what that is, sir.

3 A. Okay. Well, asbestos, for those of us who live in
4 industrialized society, we all have asbestos in our lungs.
5 It's everywhere. And with each breath we take just about,
6 unless you live in the pristine mountain environments or out
7 in the desert, chances are you are going to be breathing in
8 everyday asbestos fibers and exhale asbestos fibers. We all
9 have them inside of our lungs.

10 And we can measure how much asbestos we have by taking
11 lung tissue that has been removed from the body, either in the
12 course of an autopsy or following a surgical procedure, take
13 that to our laboratory, digesting away all the tissue. And
14 what we're left with is the residue in the lungs, the dust and
15 the debris that all of our lungs filter out. And then we're
16 able to take that into our electron microscopy lab and at very
17 high magnification examine those fibers. We're able to
18 bombard them with x-rays and tell not only if there is
19 asbestos and how much, but what type of asbestos.

20 Q. I see. As a result of all of the consultation, all of
21 the direct patient care samples you have over the years, have
22 you developed in your lab quite a database of lung burden
23 analysis?

24 A. Yes, we have.

25 Q. And have you published that and shared it in the peer

1 reviewed literature, information about that?

2 A. Yes, we have.

3 Q. And have you participated in those publications?

4 A. Yes.

5 Q. In addition, there's a book on asbestos disease called
6 The Pathology of Asbestos-Associated Diseases, and it's second
7 edition. What's your role on this book, sir?

8 A. Well, I'm one of the editors, as you can see on the front
9 piece there. And in addition to sort of reviewing what the
10 contributors had for clarity and accuracy, I wrote two
11 chapters in there myself -- no three chapters.

12 Q. Okay.

13 A. I wrote the chapters on the site of pathology of
14 mesothelioma and asbestos diseases. I wrote the chapter on
15 mesothelioma itself, and on asbestosis.

16 Q. Now, this book is in its second edition. Is there
17 another edition coming out soon?

18 A. Yes, sir. We have a third edition with Springer Verlag.
19 All the chapters are in receipt with the editorial board over
20 in Germany, and we're trying to get this out as soon as
21 possible.

22 Q. There's also a book called Malignant Mesothelioma of
23 which Andrea Tanithol (phonetic)...

24 A. Tanithol. She's a -- yeah, this is a book that was
25 published by the leadership of the German mesothelioma

1 registry and reviews in cancer research.

2 Q. And did you write a chapter in this book, sir?

3 A. I did.

4 Q. On what subject?

5 A. On the mineralogy of asbestos.

6 MR. SCHACHTER: At this point, Your Honor, we tender
7 this witness as an expert in pathology, asbestos disease, and
8 asbestos fiber type mineralogy.

9 MR. FINCH: No objection, Your Honor.

10 THE COURT: Thank you. He will be admitted as such.

11 Q. The areas I would like to elicit your opinions are
12 three -- on are three.

13 First, I'd like to get a very clear scientific
14 understanding of the mineralogical difference between the
15 various fiber types. Will you be able to provide us some
16 information on that?

17 A. I'll certainly try.

18 Q. And have you prepared some slides that might help
19 illustrate that?

20 A. Yes, sir.

21 Q. Secondly, I'd like to call upon your experience in
22 looking at so many lungs or the lung tissue from people who
23 have been in litigation to tell us what we're likely to find
24 in litigation cases. And will you be able to help us with
25 that?

1 A. I'll certainly try.

2 Q. And the last thing I'd like to elicit your opinions on is
3 whether exposure to chrysotile end products and specifically
4 gaskets and packing contribute to cause mesothelioma in
5 humans. That's sort of a subject you've written on; is that
6 correct?

7 A. I don't think I've published with that in the title, but
8 that is something that our group has looked at.

9 Q. Okay. Do you have opinions on that --

10 A. Yes.

11 Q. -- then based upon your research?

12 A. Yes.

13 Q. Okay. We'll get into that.

14 Let's start with the mineralogy of asbestos. How do we
15 divide up the mineral families of asbestos, sir?

16 A. Well, first off asbestos is not a mineralogic term per
17 se.

18 Q. Okay.

19 A. I mean, the name for minerals is usually based on --
20 either on sort of an honorific name for the individual that
21 discovered it or the properties that the mineral might have or
22 owing to the area where the fiber was found.

23 But asbestos more properly is a -- it's a commercial and
24 regulatory term. It's not something that -- people use it
25 interchangeably.

1 Q. Uh-huh.

2 A. But asbestos is more of a regulatory and classification
3 term for a group of naturally occurring minerals.

4 Q. All right. So --

5 A. And these are --

6 Q. What are the two families?

7 A. These are -- these are regulatory terms for a group of
8 very useful mineral species that have been used to antiquity.
9 You know, anywhere from the aboriginals in Scandanavia to the
10 pharaohs to the Roman times. They've been used for thousands
11 of years. And now as our mineralogists have gotten more
12 familiar with them, they've formed a classification system for
13 the actual mineral species that we now recognize are regulated
14 and classified as asbestos.

15 Q. Let me ask you this. Is it important for understanding
16 causation issues to be specific about the mineral species
17 we're dealing with?

18 A. It is. Because not all asbestos is -- this sounds sort
19 of silly to say. But not all asbestos -- they're not a group
20 of substances that have identical physical properties,
21 mineralogic and bio -- or physical chemical characteristics or
22 chemical composition.

23 Q. What are the two families, sir, so we can move on?

24 A. Okay. I'm sorry. In broadest view, the species that are
25 regulated and classified as asbestos are a group -- there's

1 the serpentine group, meaning that they have a curly,
2 snake-like appearance, and the amphibole species.

3 Q. And the ones that are used commercially purposely to make
4 products, of all the various forms, the commercial forms are
5 what, sir?

6 A. In modern times the commercial forms are amosite and
7 crocidolite. And those are fibrous forms of naturally
8 occurring fiber silicates

9 Q. And those are the amphibole commercial fiber types?

10 A. Yes, sir.

11 Q. And the serpentine commercial fiber type is what, sir?

12 A. Is chrysotile.

13 Q. Have you brought us some pictures of what these look like
14 in the macroscopic form? They're rocks, right?

15 A. Yes, they're -- yeah, they're minerals. They're
16 naturally occurring fiber silicates.

17 Q. Okay.

18 A. And chrysotile or yellow -- white asbestos is that which
19 we see on the left-hand side of our field there. Riebeckite
20 is the name of the nonfibrous form of what we recognize when
21 it's in its fibrous habit as crocidolite.

22 And amosite is the fibrous form of the mineral
23 cummingtonite grunerite. And amosite itself is also not
24 really a mineralogic term. Amosite is an acronym, Your Honor.
25 The A-M-O-S stands for the asbestos mines of South Africa. So

1 it's a species that's only found in South Africa.

2 Q. You brought us some microscopic pictures of the various
3 fiber types.

4 A. I have.

5 Q. What is this, sir?

6 A. This is a photomicrograph taken on a scanning electron
7 microscope of chrysotile fibers. Again, we mentioned these
8 are classified as serpentine, and they have a curly-Q worm or
9 snake-like appearance.

10 Q. And just so we understand the scale, what is 15um that's
11 up there. What does that mean?

12 A. That's actually, the letter right after the 50 is not a
13 U. That is a mu (phonetic). So that's 50 microns. And a
14 micron is a millionth of a meter or ten thousandth of a
15 millimeter. So again very, very -- these are taken at
16 extremely high magnifications. These are very, very small
17 fibers.

18 Q. Are the individual asbestos fibers visible to the naked
19 eye?

20 A. No, sir.

21 Q. You have a picture microscopically of what amosite looks
22 like.

23 A. Yes, sir. And by contrast, the amphiboles have a much
24 more rigid, brittle, and needle-like appearance. And that's
25 what we're seeing here with -- at our digest showing amosite

1 as opposed to the curly, pliable --

2 Q. Okay. I'm sorry to flip that.

3 And this one is crocidolite. Which family is it in?

4 A. Crocidolite resides in the same family as amosites. It's
5 an amphibole.

6 Q. And then the other -- one other amphibole that we're
7 going to talk about a little is tremolite. Is this the
8 microscopic picture of tremolite?

9 A. It is.

10 Q. And is it more similar to the chrysotile or is it more
11 similar to the amphibole?

12 A. No, you can see right here that this is much more akin in
13 terms of its growth habit to the other amphiboles, being
14 needle-like and rod shaped rather than -- rather than --
15 rather than curly.

16 Q. In terms of the crystal structure, is there a difference
17 between chrysotile on one hand and the amphiboles, amosite
18 crocidolite, and tremolite on the other?

19 A. Very much so.

20 Q. And can we demonstrate -- do you have a slide that
21 demonstrates that?

22 A. Yes, sir. May I use the...

23 Q. Yes, and if it will help to approach there so you don't
24 have to send the laser over people's heads, please come down.

25 A. I think I can maybe do this. I won't obstruct anybody's

1 view.

2 This is the crystal structure of amphiboles. And you can
3 see here in the dark gray here and in the light gray here,
4 there are two bundles of the asbestos fibers. And they
5 have -- and the asbestos fibers amphiboles are composed of the
6 silica tetrahedra, and they have on their inside a layer of
7 cations. And cations are positively charged groups of atoms.
8 And the cations are typically magnesium, calcium, iron. And
9 these are on the inside.

10 And the cations, they are on the inside. Their location
11 away from the surface confers upon them the ability to be
12 resistant to biodegradation. So when eventually these might
13 get broken down, then you see a -- can see a splitting of the
14 bundles. And then once the bundles are released, they can
15 break down into individual fibers.

16 Q. We're going to hear about a term called biopersistence.
17 Perhaps it would be helpful if you define that for us now,
18 sir.

19 A. Biopersistence is the -- it's the amount of time that an
20 inhaled particulate can persist in the body. And that is
21 dependent on a variety of different things: On the size; how
22 it gets into the body; where it gets into the body. And then
23 what it's biochemical or physical chemical characteristics are
24 will also impact on its biopersistence.

25 Q. For amphibole fibers that make it to the lung, what is

1 the biopersistence? How do we measure that?

2 A. The biopersistence of amphiboles is quite long. And
3 these often will have a residence time in the body of many
4 years. They are extremely resistant to biochemical
5 degradation. They're extremely resistant to acid
6 environments. That's what makes them such good compounds for
7 use in industrial applications is they are resistant to
8 chemical degradation. Our bodies break these fibers down
9 biochemically in the acid environment of scavenger cells. And
10 the amphiboles owing to their physical chemical confirmatin
11 are much more biopersistent.

12 Q. Biopersistence is often measured in half life. I guess
13 that means how long it takes half of them to disappear from
14 the body?

15 A. Yeah, half life is a term that is used in a lot of
16 different physical chemical settings. It's the amount of time
17 that a substance or a compound will be around. And then the
18 half life is how much time will it take until half of it goes
19 away and then another half goes away until ultimately you're
20 left with nothing.

21 Q. And is the half life of amphiboles measured in days,
22 weeks, months, decades? What?

23 A. I think the biopersistence in half life of the amphibole
24 species is measured in months to years.

25 Q. Now, the chrysotile, how does it differ and what is its

1 structure?

2 A. This is -- is what a chrysotile fiber -- cartoon of what
3 a chrysotile fiber would look like. Instead of those parallel
4 bundles, chrysotile is sort of rolled up in sheets like a
5 jelly roll or a scroll. And rather than having the cations on
6 the outside and the magnesium hydroxide groups, they are --
7 those would be on the inside and resistant to degradation with
8 the amphiboles.

9 Here we see them on the outside. And let's assume this
10 was a chrysotile fiber that found itself in the cytoplasm of a
11 macrophage. These magnesium hydroxide groups would be readily
12 leached away by the acid environment of the macrophage
13 allowing for the silica sheet to undergo degradation. And it
14 would split -- it would either become amorphous or it would
15 split into small fibrils which then would be swept away very
16 readily by the body.

17 The upshot of this is it has a much longer residence time
18 in our lungs on the orders of days to weeks.

19 Q. Okay. Much longer or much shorter time for chrysotile?

20 A. Yes, sir. The order -- the biopersistence of chrysotile
21 is much, much shorter. In fact, there are some species of
22 short fiber chrysotile like Calidria asbestos which is mined
23 out in California has the most rapid clearance times of a
24 great many different particulates.

25 Q. All right. Chemically if we were to chart out what the

1 chemical formulas of these various fibers are, have you
2 brought us a chart that will show that?

3 A. Yes, sir.

4 Q. And how does this chart work? Can you explain it to us,
5 sir?

6 A. Well, the stylized chemical formula for the amphibole
7 asbestos are the silica tetrahedra that are complexed with
8 different cations and hydroxyl groups.

9 Q. So those are the two?

10 A. Yes.

11 Q. Okay. And what is the 22?

12 A. That's the concentration of oxygen -- of the silicon
13 oxide groups here forming the central part of the core.

14 Q. So we charted out how many atoms of each of these
15 different chemicals. That will be 7 of iron manganese?

16 A. Yes.

17 Q. Is that what the MG stands for?

18 A. Yes.

19 Q. Okay. And 8 of silica and 22 of oxygen and the 2 OHs,
20 right?

21 A. Yes.

22 Q. And then crocidolite has a -- is it similar or
23 dissimilar?

24 A. Crocidolite is quite similar to amosite. The main
25 difference between that you have this sodium. The chemical

1 abbreviation for sodium is NA. So crocidolite has a sodium
2 peak on it as well.

3 Q. All right. And tremolite, is it similar or dissimilar to
4 the other amphiboles?

5 A. Yes. You can see that the -- that the central magnesium
6 of silicate core is present. Tremolite, however, the cation
7 that is complexed with it is calcium.

8 Q. Now, does this represent in the same manner the chemical
9 formula for chrysotile?

10 A. It does.

11 Q. And what is different?

12 A. Well, the predominant difference is the -- is the absence
13 of iron.

14 Q. Okay. And so in summary, would it be fair to say that
15 the amphiboles have a different chemical structural and
16 biological -- biologically relevant composition than
17 chrysotile?

18 A. Yes, sir. They have different crystalline structures
19 They have different chemical composition and different
20 physical chemical characteristics.

21 Q. Is there a biological consequence of the difference
22 between the amphiboles and chrysotile for mesothelioma
23 analysis?

24 A. Yes, there is. And I think one of the major factors that
25 influence how injurious an inhaled particle is, in addition to

1 its size and shape, is its biopersistence.

2 Q. Okay.

3 A. And the -- as we just discussed, the biopersistence
4 amphiboles is well in excess of the biopersistence of
5 chrysotile. Therefore, the amphiboles are more pathogenic in
6 terms of resulting in fibrosing disease in the lung as well as
7 malignancies, neoplasia.

8 Q. Sir, is there any question that the amphiboles, the
9 amosite, crocidolite, and tremolite, are causes for
10 mesothelioma in human beings?

11 A. Sir?

12 Q. Is there any question about whether amosite, crocidolite,
13 and tremolite is causes for mesothelioma in human beings?

14 A. Is there a question?

15 Q. Yeah. Do they cause it?

16 A. Yes. Yes, they do.

17 Q. Okay. When we discuss chrysotile, I want to be very
18 specific. Is there a distinction we should make between
19 chrysotile ore and chrysotile fibers themselves if we want to
20 be scientifically rigorous in our discussion?

21 A. I would prefer that there would be. I think it's
22 important that as we have our discussion here, we discuss
23 differences between chrysotile fibers as you might encounter
24 in a -- in a laboratory situation. I think there is a
25 difference between chrysotile and commercial products. And I

1 think there is a very real need to discuss the potential for
2 disease induction by chrysotile mine dust. I think those are
3 three separate entities and all have different abilities to
4 induce disease.

5 Q. Okay. In the context of mining populations, has the
6 chrysotile dust or the ores, or whatever is in it, in the
7 mining populations been associated with an elevated rate of
8 mesothelioma?

9 A. Yes.

10 Q. And is that attributable -- what's that attributable to?

11 A. The current feelings amongst, I think, the people that
12 have most closely evaluated this is that there's no data to
13 suggest that exposure to pure chrysotile dust without its --
14 without any type of contaminating species does not cause
15 mesothelioma. And that the remainder of the
16 chrysotile-associated mesotheliomas and asbestosis are related
17 in the presence of contaminating amphiboles.

18 Q. Okay. In the mines we've heard about fiber years as a
19 measure of how much exposure. What kinds of -- go ahead.

20 A. And I think I need -- I think I need to back up on that.

21 Q. Please do, sir.

22 A. Because chrysotile is a mineral that is mined worldwide,
23 I think in every -- every country but -- or every continent
24 but Antarctica. Chrysotile is mined in South America.
25 Chrysotile is mined in the United States of America. It's

1 mined in Canada. It's mined in South America -- or South
2 Africa. It's mined in Southeast Asia.

3 Q. All right. And do all those mining populations have an
4 increased incidence of mesothelioma?

5 A. No, they don't. And not all those mines suffer from
6 contamination of the chrysotile or with contaminating
7 amphibolic species.

8 Q. All right. Is there an area where there is a
9 contaminating amphibolic species?

10 A. Yes.

11 Q. Where is that?

12 A. That is principally in the chrysotile mining areas of the
13 province of Quebec in Canada.

14 Q. And are there some mines that do have the contamination
15 and others that don't?

16 A. Yeah. It seems to be a very geographically -- the
17 preponderance or degree of contamination with noncommercial
18 amphibole tremolite seems to be a function not only of the
19 different mine locations, but within the locations of those
20 particular mines within that area.

21 Q. Okay.

22 A. And some of the chrysotile mines in Quebec do not appear
23 to have substantial tremolite contamination at all.

24 Q. All right. At what level of exposure do you need to have
25 to -- in the mining context to the tremolite contaminated

1 chrysotile to be at risk of increased mesothelioma?

2 A. As we discussed earlier, the biopersistence of chrysotile
3 is less than the amphiboles, and that confers upon it a much
4 lower degree of pathogenicity. Therefore, it doesn't take
5 much asbestos exposure to result in the induction of diseases
6 like mesothelioma if the asbestos that you've been exposed to
7 is an amphibole like amosite or crocidolite. That's not true
8 with chrysotile. You would need to have hundreds of fiber
9 years of exposure as would typically be sustained in the
10 induction of fibrosis in the lung or asbestosis. And again,
11 we're talking in the order of hundreds of fiber years.

12 Q. All right. Of course, the issue in this case, I don't
13 believe there are any miners that have been claimants. We're
14 talking about an end product. Does the level of exposure for
15 end products like gaskets, packing, come anywhere near to the
16 levels where an increased risk from any kind of chrysotile
17 exposure has been demonstrated?

18 A. I don't believe so. Another issue that's arisen in this
19 case is the issue of idiopathic mesothelioma. Does
20 mesothelioma occur without asbestos exposure?

21 A. It can. And idiopathic is the term we use in medicine
22 where we don't know what causes a particular disease. They
23 just sort of arise sporadically within our society. Not
24 all -- we think of lung cancers as being overwhelmingly
25 associated with cigarette smoking, yet there are a handful of

1 people who don't have radon exposure, don't have any other
2 means for getting lung cancer. We look upon those as
3 idiopathic.

4 An analogous situation is within the mesotheliomas in
5 that there are mesotheliomas that -- where we can't identify
6 an exposure to asbestos. We can't demonstrate asbestos in
7 their lung tissue. And we can't come up with another
8 plausible cause for them to develop mesothelioma. There are a
9 handful of people whose mesotheliomas are neither related to
10 asbestos exposure nor idiopathic and those are rare. Those
11 are people who have undergone chest wall radiation for soft
12 tissue sarcomas. They are people who have had chronic pleural
13 effusions or fluid in the chest from tuberculosis. But again,
14 those are rare. But yes, there is an established or ballpark
15 figure of a rate of idiopathic mesothelioma in the United
16 States.

17 Q. And what is that rate just generally, sir?

18 A. Well, it has -- you have to sort of break that down both
19 in terms of topography where the mesotheliomas occur and the
20 gender. In men pleural mesotheliomas are at least 80 percent
21 related to an exposure to asbestos.

22 Q. Okay.

23 A. In women it's much less. Peritoneal mesothelioma is
24 mesothelioma that arise in the serosal membrane that invest
25 the wall of our abdomens and the visceral that are contained

1 in our abdominal cavity. Perhaps only 25 percent of
2 mesothelioma -- of peritoneal mesotheliomas in women are
3 caused by asbestos. And maybe three times that, maybe 50 to
4 60 percent in men.

5 So the association for asbestos-related mesotheliomas is
6 greatest in men in the pleura followed by greatest in men in
7 the peritoneum, and then with women bringing up the rear at
8 both sites.

9 Q. Thank you. I'd like to turn to the area of what your
10 studies have shown, your fiber burden studies have shown about
11 the nature of the asbestos found in the lungs of people, okay.

12 A. Yes, sir.

13 Q. And have you brought us information -- you've published
14 on this topic, right?

15 A. I was -- I was a co-author and my work went into this,
16 yes.

17 Q. And this is an illustration of a table that appears in
18 your article. What's the nature of that article or the name
19 of it? Just identify it if you can.

20 A. It was a case where we looked at 1,445 cases of
21 mesothelioma where we had an occupation or a reliable
22 occupation for the individual as well as a fiber burden
23 analysis.

24 Q. And were a lot of these litigation cases?

25 A. They were all litigation cases.

1 Q. All litigation cases, and primarily from defendants or
2 plaintiffs or what?

3 A. Both.

4 Q. Both. All right. And did you break down what you were
5 finding in the lungs by occupation?

6 A. Yes.

7 Q. And what were the occupation or profession categories
8 that you chose?

9 A. Well, you can see that on the bottom axis. These were
10 commercial insulators, pipefitters, boilermakers, ship --
11 people who worked in shipyards, electricians, construction
12 workers, maintenance workers at various different types of
13 factories and other concerns, as well as individuals who had
14 served in the United States navy, as well as automotive
15 workers, household contacts, and then just people who had --
16 who really didn't have an occupation that we could pinpoint.

17 Q. Okay. On this chart there is red, something that's in
18 red. What does the red illustrate?

19 A. The red illustrates what percentage of cases had
20 increased commercial amphiboles.

21 Q. Would it help if you came down here to see it or can you
22 see fine from there?

23 A. I think I'm okay.

24 Q. Okay. Great. I just want you to be comfortable. And
25 when you say increased levels, elevated levels, what do you

1 mean by elevated levels?

2 A. Well, as I mentioned, we all have asbestos in our lungs
3 assuming that we have grown up in an industrialized society.
4 And Dr. Roggli and his mentor Dr. Pratt, and others were
5 amongst the first, at least for our lab, to determine what a
6 background level of asbestos was. And that is somewhere
7 between zero and 20 asbestos bodies per gram of wet lung
8 tissue. We can -- when we measured the amount of -- uncoated
9 fibers, too, in addition to asbestos fibers.

10 A. Okay.

11 Q. We then compared what we saw amongst insulated -- the
12 insulator cohort and the pipefitter cohort and contrasted
13 those to our control population.

14 Q. Okay. So in what percentage of insulators did you find
15 an elevated level of commercial amphiboles in the lungs of the
16 people who had mesothelioma?

17 A. A hundred percent.

18 Q. For pipefitters, approximately what percent did you find
19 had an elevated level of amphiboles in their lungs?

20 A. As you can see, they are in excess of 90 percent.

21 Q. And this chart reads down similarly. Did you
22 consistently find amosite in the lungs of the people that were
23 occupationally exposed to asbestos?

24 A. Yes, we did.

25 Q. And did you draw any conclusions from that?

1 A. Well, the conclusion was that that's what caused their
2 mesothelioma.

3 Q. All right. Now, you also looked for other fiber types,
4 correct?

5 A. We did.

6 Q. Noncommercial amphiboles, that would be tremolite and
7 related noncommercial.

8 A. Yes.

9 Q. Did you ever get a consistent pattern of above background
10 levels of the noncommercial amphiboles in the majority of any
11 occupational category?

12 A. No. You can see that in -- depending on the population,
13 as high as 60 percent of electricians may have had elevation
14 of noncommercial amphiboles; whereas, automotive workers or
15 maintenance workers, there was a minority of those individuals
16 who had an increase in the noncommercial amphibole species.

17 Q. And did you also look for chrysotile?

18 A. We did.

19 Q. And did you find a consistent pattern of above background
20 levels of chrysotile in any of the occupational categories?

21 A. No. Again, you can see that there was considerable
22 variance amongst the occupational groups there.

23 Q. Now, sir, your research that you've talked to us about is
24 focused on the lung. Is the lung the place where mesothelioma
25 occurs?

1 A. No, sir.

2 Q. Then why is -- where does mesothelioma happen?

3 A. Mesothelioma typically arises in the pleura. The pleura
4 is the membrane that lines the chest cavity. And that --
5 that's called the parietal pleura and that's typically where
6 mesotheliomas originate.

7 There is also the visceral pleura which is the membrane
8 that lines the lung. But most of the mesotheliomas arise in
9 the parietal pleura. but --

10 Q. Well, then, why -- go ahead.

11 A. But the portal of entry for the asbestos fibers is
12 through inhalation; and these fibers, assuming that they are
13 of the right size and confirmation, get deposited at branch
14 points in the parts of the lung that exchange gas and then
15 they make their way out either mechanically or through
16 macrophages, macrophages being a specialized scavenger cell,
17 and these move all around the body.

18 Q. Well, why aren't you looking for the fiber burden in the
19 pleura?

20 A. Well, I think there have been studies that have shown
21 that levels of amphiboles in the pleura are predictive by
22 levels of amphiboles in the lung.

23 The other reason is we don't have control values for what
24 normal ranges of amphiboles or nonamphibole asbestos is in the
25 pleura.

1 Q. All right. And why is it important that you have a
2 control value of what the background levels are in the pleura
3 in order to use pleural tissue?

4 A. Well, if you don't know what the levels are in a control
5 population or a population that doesn't have disease, how are
6 you -- how are you going to make any sense out of a level of
7 asbestos in someone with disease?

8 Q. There is -- there's some studies published by Suzuki and
9 others that note that short chrysotile fibers are found in the
10 pleura. Are those informative or -- in proving that there is
11 causation by chrysotile of mesothelioma?

12 A. Not at all.

13 Q. Why not?

14 A. Well, I think that if you -- first off, short fiber
15 chrysotile is believed by the vast majority, I believe, of
16 scientists who work in this area as to be nonpathogenic. And
17 if you were to believe the Suzuki model where lots of short
18 fiber chrysotile is the fiber responsible for mesothelioma --
19 in the Suzuki model chrysotile would be the major cause of
20 asbestos, and I don't believe that that's true.

21 Q. The major cause of mesothelioma?

22 A. Yes, major cause of mesothelioma is amphibole asbestos,
23 not --

24 Q. And have you and your group published the view that
25 amosite exposure is the number one cause of mesothelioma --

1 A. Yes.

2 Q. -- in the United States?

3 Huh?

4 A. I don't know -- I mean, I think that is implicit in
5 several of our writings.

6 Q. Thank you. I have a picture here. What is that, sir?

7 A. Okay. This might take a little bit of orientation
8 purposes. This is a piece of -- and this is not a
9 photomicrograph.

10 This is a piece of the chest wall that has been removed
11 from somebody. We are looking at it with the naked eye. This
12 is so -- this is the inner lining of the chest wall. You can
13 sort of make out where the ribs are running. There is a rib
14 running there. And this here is the parietal pleura. And
15 what we are seeing here, these black areas here are called
16 black spots.

17 Q. Yes, sir.

18 A. And these black spots are areas where there are stomas or
19 openings in the pleura. And what is concentrating there is
20 carbon pigment. We all have carbon pigment. We have dark
21 areas in our lung tissue. We have dark areas in our pleura.
22 And that has to do with our scavenger cells having scavenged
23 black pigment that we inhale, motor vehicle exhaust,
24 industrial soot, and so forth, and it becomes concentrated at
25 those areas.

1 And Dr. Boutin, a French expert in mesothelioma, has
2 measured the amount of asbestos fibers at these areas and
3 found out that they consist exclusively of amphiboles and that
4 amphiboles are distributed heterogeneously throughout the rest
5 of the pleura.

6 This article, I think, is one of the major articles that
7 gives credence to why mesotheliomas develop here in the
8 parietal pleura. And as lung -- and as lung values are
9 predictive of pleura values, that is why we use lung tissue
10 rather than pleura.

11 Q. Now, one of the criticisms that I think is in the
12 briefing on your studies is that chrysotile, because it clears
13 much more rapidly, isn't found in fiber burden studies. So
14 that if it were causing it, your fiber burden studies would be
15 irrelevant. How do you respond to that criticism?

16 A. I've heard that before and we tend to view that as,
17 quote, the hit and run. The hypothesis is that chrysotile,
18 being a form of asbestos potentially able to cause
19 mesothelioma, goes into the body, does its work on disrupting
20 cell DNA. It's broken down by the body and then vanishes. I
21 don't think there's any scientific proof of that. And a
22 substance that doesn't reside very long in the body in my
23 opinion isn't likely to cause genetic damage, at least in a
24 real-time example within a patient.

25 Q. Now, from the studies that have now existed, do we

1 actually know the complete biomedical causation string that
2 leads from asbestos to the development of the mesothelioma?

3 A. No. And I think you bring up a -- one of -- an area in
4 biomedical science not just from mesothelioma, but for, in
5 fact, all of -- all of cancer biology. It's the steps that
6 are undertaken that result in a cell becoming transformed to
7 where a patient gets the clinical entity of having cancer,
8 that's not worked out, I mean completely worked out. There is
9 an explosion of knowledge and a lot of what are believed to be
10 individual cancers we now recognize are actually a family of
11 cancers with different mutations, different gene
12 rearrangements. Very -- very complex at the -- at the
13 molecular level.

14 Q. Do we know at the molecular level how many changes are
15 necessary in order to create a mesothelioma?

16 A. I think for solid tumors across the board, somewhere
17 between 26 and 30.

18 Q. And do we yet know which precise genetic changes have to
19 occur and in what order before you get this very rare disease
20 mesothelioma?

21 A. Maybe somebody like at Brooke Mossman's lab at the
22 University of Vermont, they might be as close to anybody to
23 knowing what all the sequences are. I don't.

24 Q. Does the fact that chrysotile in laboratory studies can
25 cause damage to a cell mean that it necessarily causes the

1 right kinds of damage that can create all of the genetic
2 mutations necessary to cause mesothelioma?

3 A. I don't believe so. And the reason for that is not all
4 experimental models of -- that result in the induction of
5 carcinogenesis have real world in vivo or in patient
6 applicability.

7 Our bodies undergo premalignant genetic changes every day
8 from cosmic radiation, from ultraviolet light, from the
9 substances that we eat, from the viruses that we are infected
10 with. So we undergo genetic damage every day. Our bodies
11 develop cancer cells or cells that are on their way to
12 becoming cancer cells every day. And fortunately, we were
13 blessed with mechanisms to repair our DNA where the genetic
14 damage occurs, and our immune system is also competent at
15 clearing those cells.

16 So even though we might generate a cancer cell, the --
17 those don't necessarily result in that particular individual
18 getting the clinical syndrome of cancer.

19 Q. One of the themes that we've seen in the briefing from
20 our learned adversaries here is that a number of public health
21 agencies have issued statements about whether chrysotile may
22 be a cause of mesothelioma. From a scientific standpoint, how
23 do you assess those in making causation decisions in an actual
24 case of whether it is a cause?

25 A. I think that scientists such as myself who work in a

1 hospital and are -- and the research that we do, we are
2 perhaps in the same church but in a different pew.

3 The organization such as -- and I assume you're talking
4 about IARC, the International Agency for the Research on
5 Cancer and other public health agencies.

6 Q. Sure. Right.

7 A. I think that public health agencies out of an abundance
8 of caution prefer to take everything back to a very
9 fundamental level and say if it could possibly cause cancer,
10 if it's been observed to cause cancer, it is best for the
11 public not to be exposed to that type of material and it's
12 best to be removed from the workplace. And that's not an
13 entirely unreasonable opinion to take if you're someone who is
14 charged with the safeguarding of the public health.

15 I think it's different, though, when you are as a
16 scientist looking at the disease in an individual case or in a
17 group of cases.

18 Q. Sir, another discrete issue that's come up is that one of
19 the IARC monographs relies heavily on a recent article by
20 Loomis and Dement that has some information about the
21 Marshville plant, and I won't belabor this because we went
22 through it a little bit with another witness. But do you have
23 any information to shed on the Marshville plant from your
24 perspective of fiber burden analysis?

25 A. My take on reviewing the Loomis and Dement article about

1 Marshville is that this was a report alleging mesotheliomas to
2 have developed in a cohort of individuals exposed only to
3 chrysotile. And there are only a handful of -- if you look
4 through the literature, there are only a handful of cohorts
5 that are alleged to be exposed solely to chrysotile, and this
6 Marshville plant was one of them.

7 And when you look at it, though, the Marshville plant
8 wasn't a plant where they used only chrysotile. They may have
9 used predominantly chrysotile. But if you go back and you
10 look at the documentation that is available for that plant
11 that was supplied by its owners, there was both amosite and
12 crocidolite used there. And my colleague, Dr. Roggli,
13 actually analyzed the lung tissue of one of the women from the
14 Marshville plant.

15 Q. What did he find?

16 A. He found amosite. And so I think that -- I think the
17 fact that chrysotile -- or crocidolite was used and amosite
18 were used at the plant, and I think the fact that amosite was
19 actually demonstrated within the lung tissue of one of the
20 workers there who developed mesothelioma, I think it
21 compromises its ability to stand as an example of a
22 chrysotile-only plant.

23 Q. Sir, you told us that in mine -- some mining populations
24 there is in excess -- chrysotile mining populations, there's
25 been an elevated risk of mesothelioma. Why doesn't that mean

1 that there must be a danger of mesothelioma from gaskets and
2 packing?

3 A. Those are two different sets of occupations with -- an
4 industrial hygienist will bear this out -- with completely
5 different exposure levels to the mineral dust. Miners and
6 millers of the mineral dust are exposed to hundreds of fibers
7 CC years. And this was looked at in -- by the workers in
8 Quebec where the women in the mining areas, the women
9 developing mesothelioma, regardless of whether they worked in
10 the factory or they just were residents of the area or had
11 exposure levels measured in the hundreds of fiber CC years.

12 Q. Sir, as a bottom line question, as an expert in asbestos
13 medicine, are you aware of any scientific -- scientifically
14 reliable methodology that would allow the formulation of a
15 reliable opinion that the exposure from gaskets and packing is
16 capable of causing mesothelioma in human beings?

17 A. I am not.

18 MR. SCHACHTER: Thank you, sir.

19 MR. FINCH: Your Honor, just give us a second to
20 switch.

21 THE COURT: Yes.

22 CROSS EXAMINATION

23 BY MR. FINCH:

24 Q. Good afternoon. Nate Finch for the asbestos claimants
25 committee.

1 Good afternoon, Dr. Sporn.

2 A. Good afternoon, Mr. Finch.

3 Q. You have testified in 45 -- 40 to 50 cases in asbestos
4 litigation, correct?

5 A. Deposition and courtroom testimony. About, yes, sir.

6 Q. Yes. And you talked on direct about how you had done
7 work with your colleague, Dr. Roggli. Isn't it true that in
8 terms of the time you've spent in testifying, 80 percent of
9 that has been for a defendant in asbestos cases, correct?

10 A. Yes.

11 Q. You have testified at the request of Garlock, obviously,
12 here today, correct?

13 A. Yes.

14 Q. You've testified at the request of Ford which made
15 chrysotile-containing brakes, correct?

16 A. Yes.

17 Q. Honeywell, correct?

18 A. Yes.

19 Q. Pneumo Abex?

20 A. Yes.

21 Q. General Motors?

22 A. Yes.

23 Q. Chrysler?

24 A. Yes.

25 Q. All those companies make chrysotile-containing brakes,

1 correct?

2 A. Yes.

3 Q. And Warren Pumps was a company that used
4 asbestos-containing gaskets and packing on or around its
5 equipment. You testified for them, too, correct?

6 A. My testimony for Warren Pumps, I believe, was not so much
7 in their defense but in insurance litigation. I wasn't an
8 expert, I don't believe, for Warren Pumps.

9 Q. You were an expert for Warren Pumps in insurance
10 litigation not in a lawsuit where a plaintiff was suing Warren
11 Pumps.

12 A. Yes.

13 Q. You're not an epidemiologist, sir, are you?

14 A. I'm not.

15 Q. You have not conducted any epidemiological -- analytical
16 epidemiological studies of asbestos and disease, correct?

17 A. I have not.

18 Q. Of your publications on asbestos, none of them
19 specifically deal with asbestos disease and the causation of
20 asbestos disease, correct?

21 A. Correct.

22 Q. You might want to speak up a little bit.

23 A. I'm sorry.

24 Q. You would agree with me that 95 percent of the asbestos
25 ever used in products in the United States was chrysotile

1 asbestos.

2 A. Yes.

3 Q. You are suspicious as to whether there is any such thing
4 as pure chrysotile outside of a laboratory setting, correct?

5 A. No, I don't know if I'm suspicious. I think that -- I
6 think there -- there is ample evidence there is pure
7 chrysotile. I mean, if I may, I think you mean pure
8 chrysotile in the absence of its natural contaminant
9 tremolite.

10 Q. Do you recall being deposed in this case, sir?

11 A. I do.

12 MR. FINCH: Would you flip up the deposition,
13 please, John. Page 39.

14 Q. "So you don't have an opinion about whether pure
15 chrysotile, hypothetically, can cause plaques or asbestos?

16 I think that -- I'm suspicious, outside of the -- of the
17 laboratory setting, whether there really does exist such an
18 entity of pure chrysotile."

19 Was that the question you were asked and the answer you
20 gave under oath in your deposition?

21 A. It was.

22 MR. FINCH: Take that down.

23 Q. You've also testified that some tremolite is unavoidably
24 retained in the milled final product, correct?

25 A. Yes.

1 Q. You would agree that asbestosis is a disease caused by
2 asbestos exposure, correct?

3 A. Yes.

4 Q. And that chrysotile fibers in suspicious doses have been
5 shown to cause asbestosis in humans, correct?

6 A. I think we have to be clear. There is a difference
7 between chrysotile fibers and chrysotile mine dust.
8 Chrysotile mine dust has been shown to cause asbestosis.

9 Q. You would agree with me that chrysotile fibers can cause
10 lung cancer.

11 A. Again, I think chrysotile -- again, we have to be very
12 specific here. Is that chrysotile fibers in animal models?
13 Yes. But if we're talking about humans, I think we have to be
14 very careful to get our terms precise and talk about
15 chrysotile mine dust.

16 Q. So it's your testimony that chrysotile from a finished
17 product cannot contribute to asbestosis in a human being;
18 that's your testimony?

19 A. Yes.

20 Q. Okay. You would agree with me that chrysotile fibers do
21 get to the pleura.

22 A. Some fibers do, yes.

23 Q. And you agree that chrysotile fibers have been shown that
24 they can damage chromosomes, correct?

25 A. Again, I think that once -- yeah, I think that all -- all

1 of these -- the first two, again, I would couch that in saying
2 is that chrysotile fibers from chrysotile mine dust -- in mine
3 dust cause asbestosis and lung cancer. Chrysotile fibers can
4 get to the pleura and chrysotile fibers in laboratories can
5 damage chromosomes.

6 Q. And they can also interfere with the body's cancer
7 defense mechanisms, correct?

8 A. Probably, yes.

9 Q. You talked about biopersistence. I believe that it's
10 your testimony -- or it's your view -- it's certainly
11 Dr. Roggli's view -- that ionizing radiation can cause
12 mesothelioma.

13 A. Yes.

14 Q. Ionizing radiation doesn't persist in the body, does it?

15 A. No, it doesn't.

16 Q. And isn't it true that cigarette smoke doesn't persist in
17 the body yet cigarettes cause lung cancer, correct?

18 A. But I think that those aren't necessarily comparable in
19 terms of what they -- how they damage or how they cause their
20 damage.

21 Ionizing radiation, I think, can cause much more severe
22 damage to the DNA of cells. I mean, ionizing radiation is
23 used to kill off the cancers. I think it causes -- and I
24 think that it's not analogous just because ionizing radiation
25 can cause genetic damage.

1 Chrysotile, which can cause genetic damage, are
2 miscreants on the same level. Certainly not everybody who
3 has -- receives ionizing radiation gets a second malignancy.

4 Q. Let's talk a little bit about tremolite and chrysotile in
5 finished products. You don't go out and test products to see
6 whether they have tremolite contamination in it, correct?

7 That's not part of what your expertise is.

8 A. No.

9 Q. And you never tested Garlock gaskets to see to what
10 extent they have tremolite in them, did you?

11 A. I didn't.

12 Q. You agree chrysotile causes mesothelioma. You testified
13 to that before without qualification, correct?

14 A. I think I have testified -- if I have said that, it's --
15 my opinion is that chrysotile mine dust causes mesothelioma.

16 Q. Do you recall testifying in a trial in the city of
17 Baltimore a few years ago in a case called Fitzgerald versus
18 AC&S?

19 A. I don't. Oh, yeah, yeah. Yeah, I do.

20 Q. And you were asked this question: "But you do agree that
21 chrysotile causes mesothelioma.

22 "Answer: I do agree that chrysotile causes
23 mesothelioma."

24 That was the question; that was your answer.

25 A. That was my answer.

1 Q. There was no qualification in that answer, correct?

2 A. No, there was not.

3 Q. You also agree that there is no safe level of exposure to
4 chrysotile fibers longer than 5 microns, correct?

5 A. Well, there -- a safe level of exposure to chrysotile
6 fibers longer than 5 microns has not been established. I
7 don't believe -- that's not the same as saying that it does
8 not exist.

9 Q. Same trial. "And you agree there is no safe level of
10 exposure to chrysotile fibers longer than 5 microns, correct?

11 "I do.

12 "I am sorry?

13 "Yes, sir."

14 That was the question that you were asked; that was your
15 answer in front of a jury in Baltimore three and a half years
16 ago, correct?

17 A. Correct.

18 Q. You would agree that a threshold level of exposure to
19 asbestos below which mesothelioma will not occur has not been
20 determined by science, correct?

21 A. It has not. But again, that does not mean that one does
22 not exist especially for chrysotile.

23 Q. You define short fiber chrysotile as fibers shorter than
24 5 microns in length, correct?

25 A. Correct.

1 Q. You have testified before that a person who has
2 mesothelioma, all of their exposures to asbestos other than to
3 short fiber chrysotile would contribute to the development of
4 the disease, correct?

5 A. Correct.

6 Q. You agree that any exposure to long fiber chrysotile
7 would pose some risk of mesothelioma.

8 A. Again, I don't know if I was given the opportunity to
9 flesh out my answer; but, yeah, if that's what you are putting
10 up from my deposition, then I must have said it.

11 Q. You would agree with me that all of these scientific and
12 research agencies have concluded that chrysotile causes
13 mesothelioma. They've all stated that in publicly available
14 pronouncements. Whether it's an IARC monograph or the World
15 Health Organization or the United States Surgeon General, all
16 of those agencies on that chart have said that chrysotile
17 asbestos causes mesothelioma in human beings.

18 A. Yes, they have.

19 Q. Okay. Let's go -- you talked a little bit about public
20 health agencies and regulatory purposes. You would agree with
21 me that the International Agency for Research on Cancer
22 doesn't regulate cancer. It tries to establish what is a
23 confirmed human carcinogen, correct?

24 A. That's what they say.

25 Q. They don't pass laws or regulations or rules to try to

1 reduce carcinogens or the epidemic of cancer in the world.

2 They look at what causes cancer, right?

3 A. They look at what they think causes cancer or what they
4 have written causes cancer.

5 Q. Okay. The National Cancer Institute, likewise, doesn't
6 regulate cancer in the United States and it has stated that
7 chrysotile causes mesothelioma, correct?

8 A. I've not seen that.

9 Q. You don't know one way or the other, correct?

10 A. I don't.

11 Q. All right. Did Garlock ever show you material data
12 safety sheets that it put out for its chrysotile sheet
13 gaskets?

14 MR. SCHACHTER: Your Honor, we have to object to the
15 introduction of the Garlock MSDS. Subsequent remedial
16 measures are not admissible to prove liability.

17 MR. FINCH: Your Honor, this is not -- first of all,
18 this is not a liability issue in this case.

19 Secondly, it's not a subsequent remedial measure.
20 There were plenty of people that were exposed to Garlock
21 gaskets after the date of this document.

22 It's an admission by Garlock as to what working with
23 its gaskets can do. An MSDS sheet doesn't go to end users.
24 It's a document that's created for -- for -- by Garlock and it
25 doesn't -- it's not like a label on a gasket or it's not like

1 a label that goes to the end using public. It's sent out by
2 Garlock and it's an admission on the question of causation,
3 but it is -- it is not a subsequent remedial measure.

4 MR. SCHACHTER: If I may, Your Honor, just briefly.

5 As we briefed in our Daubert briefing, MSDS has been
6 uniformly rejected as proof of causation. These are
7 required documents under government regulations requiring to
8 be protective and over -- airing on the side of over
9 protection just like all other protective measures. And we
10 therefore object to the use of them.

11 MR. FINCH: Your Honor, at the time this document
12 was written, the government did not require the exact language
13 Garlock put in its MSDS.

14 THE COURT: We'll admit the document and let you
15 examine about it.

16 MR. FINCH: Thank you. And there is an exhibit
17 number. I will give you the exhibit number when we offer it
18 in our case, but I just want to show it to Dr. Sporn.

19 Q. This is a document, chrysotile sheet test MSDS written by
20 Garlock. It talks about chrysotile asbestos in the gaskets.
21 "What are the health hazards? Chronic breathing of amounts of
22 asbestos fibers can cause lung disorders such as asbestosis,
23 pleural plaque, lung cancer, and mesothelioma.

24 "Dust from sheet should be treated as free asbestos.
25 Secure the area. Clean up using HEPA-filtered vacuum or wet

1 sweep. Do not clean up in a method that creates dust."

2 That's what Garlock said in its MSDS. It never showed
3 that to you, Dr. Sporn?

4 A. No.

5 Q. You talked a little bit about Calidria asbestos on direct
6 examination. Do you recall that?

7 A. Yes.

8 Q. That's made by a company called Union Carbide and the
9 Calidria Corporation, right?

10 A. Correct.

11 Q. Has Union Carbide ever showed you its MSDS on Calidria
12 asbestos?

13 A. No.

14 Q. "Over exposure to chrysotile asbestos has caused damage
15 to lungs, (asbestosis), lung cancer and mesothelioma of the
16 pleura and peritoneum. Symptoms, which are usually not
17 manifested until 15 to 20 years after exposure, include
18 labored breathing, chest pains, weakness, and chest
19 tightness."

20 That's what Union Carbide has said about its Calidria
21 asbestos. They never showed that to you?

22 A. No.

23 Q. At the end of my direct exam -- my cross exam, I'm going
24 to ask you some questions about the health impacts on people
25 with mesothelioma. So we'll get to that in a little bit. But

1 I want to talk to you a little bit about your fiber burden
2 studies.

3 The -- you acknowledged on direct that fiber burden
4 analyses look at what's found in the lung, correct?

5 A. Correct.

6 Q. And not what's found in the pleura, correct?

7 A. Correct.

8 Q. And that mesothelioma obviously occurs in the pleura,
9 correct?

10 A. Correct.

11 Q. All right. Now, your case series of 1,445 cases that you
12 and Dr. Roggli are authors of, that was -- that was published
13 in the peer reviewed literature, correct?

14 A. Yes.

15 Q. And while it's not an analytical epidemiology study, it's
16 a case series that you at least rely on to inform your views
17 about the causation of mesothelioma, correct?

18 A. Correct.

19 Q. And you would agree with me that by 1960, the world came
20 to the conclusion that there was -- that crocidolite asbestos
21 could cause mesothelioma based on a paper published by Wagner
22 in 1960, right?

23 A. Correct.

24 MR. SCHACHTER: Objection, Your Honor. He's going
25 into the history of mesothelioma which is beyond the testimony

1 of the other witnesses.

2 THE COURT: We'll let him answer the question if he
3 can. Overruled.

4 A. Repeat, please.

5 Q. My question was the world literature, the medical and
6 scientific community in 1960 concluded that crocidolite
7 asbestos could cause mesothelioma based on a seminal paper by
8 Dr. Wagner in South Africa which was published in 1960,
9 correct?

10 A. Yes.

11 Q. Is that yes?

12 A. Yes.

13 Q. Okay. That was a case series. That wasn't an analytical
14 epidemiology study, correct?

15 A. Yes.

16 Q. Okay. Now, on the question of fiber burden analysis,
17 this is an article that you and Dr. Roggli published together
18 with Vollmer and Kelly Butnor in 2002 called "Tremolite and
19 Mesothelioma."

20 A. Right.

21 Q. And it discusses the presence of tremolite in people's
22 lungs. And the group of people that you're looking at is a
23 subset of your 1,445 case series; is that correct?

24 A. Right. Yes.

25 Q. Okay. And am I correct in your 1,445 patient -- or

1 litigation case series, you only had lung tissue to do fiber
2 burden analysis in about 280 people.

3 A. I can't remember the exact number that we had.

4 Q. It was less than 300 though, right?

5 A. Yes.

6 Q. Okay. And of that -- so only about a fifth of them did
7 you have any kind of lung tissue to do a fiber burden analysis
8 on.

9 A. Right.

10 Q. And that's pretty common in asbestos litigation. A lot
11 of times you don't have lung tissue because you're only going
12 to get it if the person dies and there's an autopsy or if
13 there is an extrapleural pneumonectomy, right?

14 A. Correct.

15 Q. So a lot of times you'll have a patient with mesothelioma
16 who might testify at a trial or might be alive or they die and
17 for whatever reason there wasn't an autopsy so you don't have
18 the lung tissue to do the fiber burden analysis, right?

19 A. Right.

20 Q. And of that 280 odd cases where you did have the lung
21 tissue to do the fiber burden analysis, only ten of them were
22 brake workers, right?

23 A. Yes.

24 Q. So when you showed the judge the chart with the
25 occupations and what percentage of them had commercial

1 amphiboles in their lungs and you had the brake workers, you
2 had a heck of a lot more insulators than you had brake
3 workers. You only had ten brake workers to look at, right?

4 A. Right. And that's because this is not a common disease
5 amongst brake workers. This is a common disease amongst
6 insulators.

7 Q. Let me -- in your paper, 2002 paper, Roggli, Sporn -- I'm
8 cutting out the middle two authors to make it fit on the
9 screen, but you know you had two co-authors.

10 A. Butnor and Vollmer.

11 Q. No disrespect to Butnor and Vollmer, but to make it
12 simple, you recognize that as a statement out of your paper,
13 correct?

14 A. Yes.

15 Q. Okay. And what you wrote along with Dr. Roggli is that
16 "although chrysotile concentrations were also strongly
17 correlated with tremolite fiber burden, chrysotile accounted
18 for a smaller percentage of the tremolite deviance. In this
19 regard it should be noted that chrysotile does not accumulate
20 in lung tissue samples to the extent that the amphiboles do,
21 as it is broken down into smaller fibrils that are more
22 readily cleared from the lungs. These smaller fibrils would
23 have been missed by our technique."

24 And you're talking about your technique that your
25 laboratory at Duke uses, correct?

1 A. Yes.

2 Q. "Since we only counted fibers that were greater than 5
3 microns in length. Furthermore, chrysotile fibers split
4 longitudinally."

5 That means they split long ways, right?

6 A. Yes.

7 Q. Okay. "In vivo."

8 That means in the living animal, correct?

9 A. Right.

10 Q. Okay. "To produce long fibers that are less than 1
11 micron in diameter."

12 And so what you mean in there, you can have chrysotile
13 fibers that are longer than 5 microns long, but they're too
14 thin to see by your microscopic techniques because they're
15 less than 1 micron in diameter, correct?

16 A. Correct.

17 Q. All right. "Such long, thin fibers would likewise be
18 missed at the screening magnification that we use. Since
19 chrysotile content is poorly detected by SEM," that's scanning
20 electron microscopes, "and fiber burden is a poor indicator of
21 total chrysotile exposure, other information must be sought to
22 address this question.

23 "The best indicator of chrysotile exposure is an
24 occupational history."

25 That's what you wrote in 2002, correct?

1 A. Correct.

2 Q. You're familiar that other people have written papers on
3 the limitations of drawing etiologic inferences based on
4 measurement of asbestos fibers from lung tissue, correct?

5 A. I've not seen this particular one. I've heard that.

6 Q. Okay. So you're not familiar with -- you're familiar
7 with Irving Selikoff, correct?

8 A. Sure.

9 Q. He published a book called "The Third Wave of Asbestos
10 Disease" in about 1990 or 199 -- early 1990s. Do you remember
11 that?

12 A. I've never read it.

13 Q. Okay. So you would never have seen this paper by Dean
14 Baker where he goes through all the limitations on why fiber
15 burden analysis isn't a good indicator of what someone was
16 exposed to or whether the dose that matters is what they were
17 exposed to. You can't comment on that.

18 A. Well, I -- as I said, I've not read it. But I would -- I
19 think that a limitation and not identifying a fiber that we
20 have now come to understand is not likely pathogenic, that's
21 not particularly important.

22 MR. SCHACHTER: Your Honor, object to his displaying
23 and cross examining on an article that he hasn't --

24 MR. FINCH: Okay. If the witness isn't familiar
25 with it, I'll move on.

1 Q. You would agree with me that we've already said that
2 the -- there are -- some of the limitations on fiber burden
3 analysis, you're looking at the lung not the pleura, correct?

4 A. Right.

5 Q. You're looking at less than 1 percent of the lung tissue
6 when you do a fiber burden analysis, correct?

7 A. Correct.

8 Q. You have to sample a portion of the lung so there are
9 going to be different levels of asbestos fibers found in the
10 lung depending on where the sample hits, correct?

11 A. Well, yeah, but for that reason we tend to use the
12 peripheral lung where the asbestos fibers tend to be
13 deposited.

14 Q. All right. We -- your 2002 article mentioned the
15 microscope magnification/resolution issue, correct?

16 A. Yes.

17 Q. Meaning that your microscopes that you use at Duke don't
18 see really thin fibers. By that I mean fibers less than 0.15
19 microns. You don't crank up the magnification high enough to
20 see those thin fibers, right?

21 A. Yeah. Why would we? I mean, they're not pathogenic.

22 Q. There's also the issue of -- we talked about on direct
23 the biopersistence of different fiber types, right?

24 A. Yes.

25 Q. That's a limitation on the use of what's in somebody's

1 lungs to make any kind of statement of what they were exposed
2 to 30, 40, 50 years ago, correct?

3 A. Correct.

4 Q. You agree that chrysotile asbestos when breathed into the
5 lungs can cause or contribute to a carcinogenic process prior
6 to degrading.

7 A. No. And again, I agree that chrysotile mine dust with
8 its natural contaminant can. This seems -- this particular
9 slide that you're showing me seems to me to be a restatement
10 of the -- what we call the hit and run process. The
11 chrysotile is breathed into his lungs -- is breathed into an
12 individual's lungs. Contributes to a carcinogenic process and
13 it disappears. And for me I don't know of any scientific
14 basis to support that.

15 MR. FINCH: Can you show the deposition of Dr. Sporn
16 taken June 10, 2008, in a case called Depiece versus Riley
17 (phonetic).

18 Q. Dr. Sporn, you were asked this question: "Do you have an
19 opinion as to whether chrysotile asbestos when breathed into
20 the lungs can cause or contribute to a carcinogenic process
21 prior to degrading?

22 "Yes.

23 "And what is your opinion?

24 "It can.

25 "It can, is that what you said?

1 "Yes."

2 Is that your answer under oath in 2008?

3 A. That was.

4 Q. All right. You would agree that the world's scientific
5 literature would treat this as a chrysotile cohort. Something
6 that is -- predominant chrysotile exposure with potential or a
7 little bit of amphibole exposure.

8 A. I'm sorry, you're going to have to --

9 Q. Sure. If there was a cohort of people that were exposed
10 predominantly to chrysotile, 99 percent to chrysotile --
11 95 percent to chrysotile, and there might have been some
12 amphibole contamination, either through the tremolite in the
13 ore or because there might have been some amphibole
14 contamination in another part of the plant, the world's
15 scientific literature in peer reviewed publications treats it
16 as a chrysotile cohort.

17 A. I'm only aware of maybe two or three people that would --
18 cohorts that would fit into that definition.

19 Q. Okay. Are you familiar with this paper that was
20 published in the British Journal of Cancer in 2012 called
21 "Estimating the Asbestos-Related Lung Cancer Burden from
22 Mesothelioma Mortality." The lead author is McCormack, and
23 Julian Peto is one of the co-authors.

24 A. Yes.

25 Q. Okay. And they list in that paper chrysotile cohorts,

1 and they called them pure or predominant. And they go through
2 15 different studies and they treat those studies -- they call
3 them chrysotile cohorts for the purposes of the analysis they
4 are doing in this peer reviewed paper in the British Journal
5 of Cancer, correct?

6 A. Yes.

7 Q. Okay.

8 A. But if you could go back to that slide, I would point out
9 that I would disagree with number 20, the Balangero Mine. And
10 I would disagree with the North Carolina textile plants which
11 we have already been through in our discussion today.

12 Q. Okay. You would disagree with Julian Peto, and the other
13 people, who is a world-renowned epidemiologist in calling this
14 a -- these are chrysotile pure or predominantly chrysotile
15 cohorts.

16 A. I would disagree with the -- actually, I would also
17 disagree with him on number 21, the Quebec asbestos factory.
18 That was not -- that was not a pure chrysotile plant. There
19 was amphibole asbestos that was demonstrated in the lungs of
20 patients with mesothelioma.

21 I would also disagree with 20. I would also -- the
22 Balangero Mine.

23 And I would also disagree with 27, the North Carolina
24 textile plants in the USA.

25 Again, they're saying predominantly chrysotile. When

1 they say predominantly chrysotile, it sounds to me like
2 there's a difference between predominantly chrysotile and pure
3 chrysotile. I think in a predominantly chrysotile plant that
4 there was an amphibole asbestos used in those facilities.

5 Q. And these are the cohort studies that IARC and the
6 National Academy of Sciences and the United States NIOSH rely
7 upon in doing risk assessments for the relative potency of the
8 different fiber types. Those studies have been published in
9 the peer reviewed literature.

10 A. I'm not sure they seek to establish fiber potency. But I
11 think that they certainly do use these as a means for
12 determining that chrysotile causes asbestos, which is why I
13 think the IARC document is flawed.

14 Q. You just said chrysotile causes asbestos. I think you
15 meant to say chrysotile causes mesothelioma.

16 A. I beg -- yeah, chrysotile mine dust causes mesothelioma.

17 Q. The world scientists outside of the courtroom debate, for
18 whatever purposes they put it to, whether you think it's
19 overly precautionous or not, rely on these cohorts to make a
20 conclusion that chrysotile causes mesothelioma.

21 A. Well, the world -- the world opinion is also -- IARC
22 relies on the opinions of people like Infante and Phil
23 Landergan who are very much a part of the litigation.

24 Q. This was a letter to the editor published a couple of
25 weeks ago by Dr. McCormack and Dr. Peto. And what they write

1 is "On the carcinogenicity of chrysotile, our article clearly
2 shows that there are both excess of mesothelioma (four
3 mesothelioma deaths per one thousand deaths) and lung cancer
4 associated with chrysotile. This is entirely consistent with
5 the IARC classification of chrysotile as a group 1 carcinogen
6 to humans. At no point do we conclude that mesothelioma
7 occurring in chrysotile exposed cohorts is due to other
8 asbestos types; rather, we consider it valid to discuss that
9 when multiple carcinogenic fibers are present. The relevant
10 contribution of each is more difficult to disentangle. This
11 is particularly the case for chrysotile in the presence of
12 amphiboles because, as concluded by the most recent meeting of
13 the IARC monographs, the latter appears to have a greater
14 potency for the induction of mesothelioma than does
15 chrysotile."

16 You're familiar with Dr. Peto's statement to that effect,
17 correct?

18 A. Yes.

19 Q. Let's talk a little bit about fiber potency differences.
20 You're aware that --

21 MR. SCHACHTER: Again, Your Honor, this goes beyond
22 the direct examination. We didn't talk about potency
23 differences at all.

24 THE COURT: All right. Go ahead.

25 MR. FINCH: He talks about it in his report.

1 THE COURT: Go ahead.

2 Q. You're aware that in 19 -- in 2008 the Environmental
3 Protection Agency convened a science advisory board to look at
4 a fiber potency model proposed by Berman and Crump?

5 A. Right.

6 Q. That estimated the relative potency of the various fiber
7 types, crocidolite, amosite, chrysotile, correct? You're
8 aware that happened.

9 A. Yes, I am.

10 Q. And you're aware that your colleague, Victor Roggli,
11 wrote a letter on behalf of Caterpillar to the EPA science
12 advisory board that says there is a big difference between the
13 fiber types, correct?

14 A. Correct.

15 Q. And you're aware that the science advisory board included
16 people like Julian Peto on it, correct?

17 A. I don't know who sat on the EPA on this regard.

18 Q. All right. You do know that the EPA convened a special
19 panel of experts from around the world to analyze this
20 question, right?

21 A. Yeah, and I know that they chose to ignore what was put
22 forth, recommended to them by Berman and Crump.

23 Q. They chose to say -- okay, that's fine.

24 Ultimately, the EPA science advisory board concluded
25 there wasn't enough historical exposure data to quantify the

1 differences between asbestos fiber varieties. Isn't that what
2 they concluded?

3 A. That's sort of the summation of what they concluded.

4 Q. Okay. Now, the judge is going to hear testimony about
5 projections of mesothelioma deaths done by doctors at Mt.
6 Sinai in 1982 as a way to give him a basis to project the
7 future number of asbestos claims in this case. You're
8 familiar with Nicholson, Perkel, Selikoff, "Occupational
9 "exposure to asbestos: projected mortality and population at
10 risk," correct?

11 A. Yes.

12 Q. Okay. You're aware that Dr. Nicholson and Dr. Selikoff
13 have basically gotten that projection right over the past 30
14 years, correct?

15 MR. SCHACHTER: Objection, beyond his area of
16 expertise.

17 THE COURT: Well, he can answer it if he can.

18 A. I can't answer that.

19 Q. So you don't know whether -- you're certainly not going
20 to dispute if other people come in here and testify that
21 Nicholson got it right, you're not going to say he didn't get
22 it right, correct?

23 A. No, I'm not -- I'm completely neutral in that regard.

24 Q. Okay. You recognize William Nicholson is an eminent
25 scientist in the field of asbestos-related disease, correct?

1 A. I'm not familiar with much of his writings at all.

2 Q. You're not aware that Nicholson has published multiple
3 papers on the epidemiology of asbestos-related diseases?

4 You're not familiar with that?

5 A. I'm sure I've seen them. I can't -- I can't cite one
6 offhand.

7 Q. Okay. So I guess -- are you familiar at all with Dr.
8 Nicholson's paper, "The Carcinogenicity of Chrysotile
9 Asbestos," a review that he published in 2001, one of the last
10 things he published in the peer reviewed literature before he
11 died?

12 A. I'm not aware of that.

13 Q. Don't you cite that paper in your rebuttal report in this
14 case? Or at least you comment on Dr. Welch's citation to it.

15 A. I may have, yes.

16 Q. Okay. And what Dr. Nicholson, who 30 years ago projected
17 mesothelioma deaths in the United States and got it right.
18 What he wrote in 2001, "The case that chrysotile is a potent
19 causative factor in producing mesothelioma is a strong one.
20 It is shown to be so in a comparison of more than 40 studies
21 of different fiber exposure circumstances.

22 "All available data suggests that it dominates the risk
23 in those circumstances where it is the principal fiber used.
24 The risk of chrysotile in producing mesothelioma is similar to
25 that of amosite on a per fiber exposure basis."

1 He published this in the peer reviewed literature in
2 2001, right?

3 A. He may have, but he didn't get that right.

4 Q. Okay. You're saying he got that wrong.

5 Well, you're familiar -- we just talked about this paper
6 from the British Journal of Cancer six months ago, right?

7 A. Right.

8 Q. Okay. There they are estimating asbestos-related lung
9 cancer burden from mesothelioma mortality. And they've got
10 chrysotile cohorts. They've got crocidolite cohorts. They've
11 got three or four amosite cohorts. And they've got a lot of
12 mixed cohorts. You're familiar with that paper, correct?

13 A. Yes.

14 Q. All right. And they have a table that compares
15 mesothelioma deaths per 1,000 non-asbestos related deaths,
16 right, Dr. Sporn?

17 A. That's what that table is.

18 Q. All right. And so the biggest rate of mesothelioma
19 deaths per 1,000 non-asbestos related deaths is for
20 crocidolite at 93.2, right? That's what you would expect.

21 A. Sure.

22 Q. Second biggest is mixed, right? Mixed fibers exposures
23 is at 42 per 1,000, correct?

24 A. Yes.

25 Q. Then the next one down is amosite. That's at 18 per

1 1,000 non-asbestos related deaths, right?

2 A. Right.

3 Q. And then when you get to chrysotile, it's down to 4.1,
4 right?

5 A. Right.

6 Q. Okay. So if you do the ratios, 93 to 18 to 4 is about
7 like 100 to 20 to 4, right?

8 A. Close enough.

9 Q. Which is more like 25 to 5 to 1, right?

10 A. More or less, yes.

11 Q. All right. Latency period. You would agree with me that
12 the median latency period for mesothelioma in your paper, the
13 1,445 paper, was on the order of 30, 35 years, right?

14 A. It's measured in decades, yes.

15 Q. Okay. But if I were to say 35 years is the median
16 latency period for mesothelioma in the United States, you
17 wouldn't quarrel with that.

18 A. No.

19 Q. Okay. And what that means, that means that half of the
20 cases would have -- by latency, just so we're real clear.
21 Latency is the time from the first exposure to asbestos until
22 someone develops mesothelioma however many years later,
23 correct?

24 A. Latency periods are the time from the first exposure to
25 anything to the development of clinical disease, yes.

1 Q. So if we're talking about cigarette smoking, if you first
2 started picking up cigarettes when you were 15 and you
3 developed lung cancer at 65, that's a latency period of 50
4 years. That's what you mean by latency in the medical
5 epidemiological context, right?

6 A. Right.

7 Q. Okay. So when you're talking about median latency, that
8 means that half the people would have a latency -- in your
9 group would have a latency shorter than 35 years and half of
10 them would have it more than 35 years, correct?

11 A. Right.

12 Q. Okay. And although the median is 35 years, it can be --
13 there are cases in your case series where the first exposure
14 was 50, 60 years before, correct?

15 A. Absolutely.

16 Q. Okay. And you would agree with me that when you're --
17 the people in your 1,445 case series, a lot of times they
18 often don't know all the asbestos they may have been exposed
19 to in their career or their life, correct?

20 A. Yes.

21 Q. And a lot of times the patient has got mesothelioma,
22 patients you have treated with mesothelioma will say, yes, I
23 worked around this kind of asbestos product, but they won't --
24 and they will have forgotten about or they won't have
25 remembered that they were exposed to asbestos in the navy ten

1 years before, right?

2 A. They might not have even known.

3 Q. They might not even know. And it's only when you look in
4 some of their lungs you say, hey, jeez, this guy was exposed
5 to asbestos from pipe insulation in the navy; therefore, there
6 was some exposure that he totally honestly with you didn't
7 know anything about, right?

8 A. Sure.

9 Q. So if somebody says, you know, I don't know if I was
10 exposed to asbestos from insulation in the navy, they're not
11 lying. That's perfectly plausible, correct?

12 A. Sure.

13 Q. And you would agree with me on this concept of latency,
14 if you were exposed to asbestos five years ago and you got
15 mesothelioma, that's not within the latency period that you
16 would expect to be an asbestos-associated mesothelioma,
17 correct?

18 A. Latency periods of less than a decade have been described
19 usually only in cases of massive exposure.

20 Q. And what I'm getting at is if the median latency period
21 for mesothelioma is 35 years, which means half the cases would
22 have latency periods shorter than that and half longer than
23 that, if you only waited 30 years after a cohort of people was
24 exposed, you wouldn't necessarily know if mesothelioma was
25 going to develop in that cohort, correct?

1 A. No. There's a lot of things you don't know. I mean, you
2 don't know who -- I mean, that's the big mystery with
3 mesothelioma. We have thousands and thousands and thousands
4 of people who have been exposed to asbestos and it's a rare
5 disease. There are 3,000, 4,000 cases in our country a year
6 and hundreds of thousands of people have been exposed. So I'm
7 not sure what that means.

8 Q. What I'm getting at is, let's say you design a cohort
9 study where you're looking to see whether exposure to a
10 carcinogen causes a disease and you know the latency period,
11 the average latency period for the disease causation is 30
12 years and you have a cohort of a thousand people and you
13 expose them all to the same amount of stuff. If you only wait
14 20 years, you're not going -- it's not going to tell you
15 anything really about whether or not that carcinogen can or
16 cannot cause a disease, correct?

17 A. You don't know what --

18 Q. Okay.

19 A. You don't know what -- you don't know anything one way or
20 another. You don't know that these folks are definitely going
21 to come down with mesothelioma. You only can deal with the
22 data that you have at hand.

23 Q. Okay.

24 A. And I would submit to you that, you know, to speculate
25 beyond who might have got mesothelioma, that lacks scientific

1 foundation.

2 Q. I'm not trying to get you to speculate, Dr. Sporn.

3 Let me ask you this. Are you familiar with the preamble
4 to the IARC 2012 monograph where they say that "it is
5 important to note that evidence of lack of carcinogenicity
6 obtained in this way from several epidemiological studies can
7 apply only to the types of cancer studied, the dose levels
8 reported and to the intervals between first exposure and the
9 disease onset observed in these studies. Experience with
10 human cancer indicates that the period from first exposure to
11 the development of clinical cancer is sometimes longer than 20
12 years. Latent periods substantially shorter than 30 years
13 cannot provide evidence for lack of carcinogenicity."

14 That's what IARC said in the preamble in 2012, correct?

15 A. That's what they've stated there.

16 Q. Okay. You don't disagree with that, correct?

17 A. Well, it's a very bland statement. It also -- I would
18 also add if I was penning that, I would say latent periods
19 substantially shorter than 30 years cannot provide evidence
20 for lack of carcinogenicity nor can they provide meaningful
21 information regarding the probability of the development of
22 carcinogenicity 10, 20 years down the road.

23 Q. Isn't it true that none of the brake worker studies that
24 Dr. Garabrant talked about had a latency period for all the
25 people in the cohort longer -- all of them had longer than 30

1 years?

2 A. I don't remember what the latency periods were.

3 Q. All these scientific agencies have said --

4 A. But if you'll excuse me. The latency period also is
5 inversely proportional to the degree of exposure.

6 Q. I'll get to that in a minute. What that means is the
7 more asbestos you're exposed to, the shorter the duration for
8 the latency period, correct?

9 A. Correct.

10 Q. So if you're exposed to a ton of asbestos, you might have
11 a 20 year latency period; less asbestos exposure you might
12 have 50 years?

13 A. If you get mesothelioma at all.

14 Q. If you get it at all, right.

15 A. But you may not. And the minority of people do get
16 mesothelioma.

17 THE COURT: Let's take a break.

18 MR. FINCH: Okay.

19 THE COURT: Come back, I guess, at 10 after.

20 (Brief recess at 3:57 p.m.)

21 THOMAS SPORN

22 CROSS EXAMINATION (Cont'd.)

23 BY MR. FINCH:

24 Q. Good afternoon, again, Dr. Sporn.

25 A. Mr. Finch.

1 Q. I think I'm in about the 7th or 8th inning here, so we're
2 going to get you out of here.

3 You would agree with me that all of these organizations
4 have stated that there is no safe level of exposure to
5 asbestos.

6 A. Yes.

7 Q. And they haven't made any qualification or exception for
8 chrysotile asbestos in their statements, correct?

9 A. No, sir, they haven't.

10 Q. Are you familiar with the National Cancer Institute, if
11 you go on the internet today they have something called a fact
12 sheet relating to asbestos exposure and cancer risk. You
13 Google that, National Cancer Institute fact sheet, you'll find
14 that on the internet, right?

15 A. No doubt.

16 Q. And you're aware that the car companies wrote to the
17 National Cancer Institute and sent them a bunch of
18 epidemiological studies sometime in 2005 or 2006 and asked
19 them to change the National Cancer Institute fact sheet,
20 correct?

21 A. I'm not aware of that.

22 Q. Well, today the National Cancer Institute fact sheet
23 states, "Studies evaluating the cancer risk experienced by
24 automobile mechanics exposed to asbestos through brake repair
25 are limited, but the overall evidence suggests there is no

1 safe level of asbestos exposure." And they cite to a couple
2 of documents, correct?

3 A. That's what they say there.

4 Q. And then one of the things they cite to is the World
5 Health Organization 1998 publication on chrysotile asbestos
6 which is about 300 pages thick, correct?

7 A. I'm not sure about that. But as far as the statement
8 you're displaying in front of me, I would submit to you that
9 there are studies evaluating the cancer risk experienced by
10 automobile mechanics exposed to asbestos in brake repair are
11 not limited, but there are a number of them. And that the
12 overall evidence suggests there is no safe level of asbestos
13 exposure. Now are they talking about through individuals
14 engaged in brake repair, in which case I would disagree. If
15 they were talking about safe levels of asbestos exposure in
16 general, that's a reasonable point.

17 Q. Let's talk about black spots. You're familiar with the
18 Journal of Respiration?

19 A. Yes.

20 Q. Okay. This is an article published in the Journal of
21 Respiration in the year 2002 that discusses the "Black Spots
22 of the Parietal Pleura: Morphology and Formal Pathogenesis."
23 Do you see that article?

24 A. Yes, I do.

25 Q. What they said is that black spots seen as

1 flat-to-nodular lesions of the parietal pleura are common
2 findings in former miners. They represent areas of coal dust
3 accumulation."

4 The conclusion -- and this is in the abstract of the
5 paper. "Although there are hints for an increased
6 proliferation of mesothelial cells in some areas with black
7 spots, our findings do not support the classification of black
8 spots as an obligate early lesion in the development of
9 malignant mesotheliomas."

10 Do you see that?

11 A. I do. And I don't know if you care to ask me what I
12 think of this, but I think this is -- it is an article that
13 flies in the face of something that I referred to in my report
14 by Dr. Boutin and colleagues where black spots seen as
15 flat-to-nodular lesions of the parietal plural are common
16 findings not just in former miners, but they are common
17 findings in the population at large and people who live in
18 areas where we breathe a lot of soot and automobile exhaust,
19 they correspond to stomas. And that's where amphiboles
20 congregate.

21 And I would disagree, although I have not read this
22 paper, I would disagree with the -- "although there are hints
23 for an increased proliferation of mesothelial cells in some
24 areas with black spots, our findings do not support the
25 classification of black spots as an early lesion in the

1 development of mesotheliomas."

2 I don't think that's what Boutin and colleagues were
3 saying. I think they were saying -- and their bottom line in
4 their paper is that's why mesotheliomas begin in the parietal
5 pleura and not in the visceral pleura of the lung.

6 So I think this is all well and good, but I don't think
7 that this changes my opinions regarding the translocation of
8 amphibole fibers to the pleura nor the cite of development of
9 malignant mesothelioma in the parietal pleura of the chest
10 wall.

11 Q. Let me see if I can boil all that down. You would agree
12 that this article is inconsistent with Boutin's conclusion
13 about the significance of black spots.

14 A. Well, I don't think Boutin was trying to say that the
15 black spots were precursor lesions, which they refer to as
16 obligate early lesions in the development of mesotheliomas.
17 All that Boutin was saying is the commercial amphibole fibers
18 are concentrated in the black spots in the parietal pleura.
19 Parietal pleura is where the vast majority of mesotheliomas
20 develop and that's a potential explanation for why.

21 Q. A potential explanation.

22 A. Well, a very good explanation why.

23 Q. But you would agree with me there are scientists who
24 differ with you about the significance of black spots in the
25 parietal pleura.

1 A. Well, this is the first one I've seen and -- I know a
2 great many pathologists from Germany and I don't know any of
3 these individuals and so I'm not sure what their experience
4 with mesothelioma is so -- I mean, the paper is what it is,
5 but this doesn't, I don't think, change the contravening
6 wisdom regarding development of --

7 Q. It was -- it was published in a peer reviewed journal
8 called Respiration, correct?

9 A. Yes.

10 Q. All right. You talked a little bit about exposure to
11 asbestos from ambient air. I just want to see if you'll agree
12 with some basic third grade math.

13 You would agree a reasonable approximation, if somebody
14 is working, how many breaths of air they take in a minute is
15 anywhere between 12 and 20 breaths. Probably around 16 if
16 you're an adult man and you're not some four minute miler or
17 something, right?

18 A. Yeah, I mean, tidal respiration is 12 to 16 breaths per
19 minute.

20 Q. And one breath of air is approximately 500 cubic
21 centimeters that go into the lung that is not recycled air but
22 new air into your lung.

23 A. Tidal -- your tidal volume, your tidal breathing is based
24 probably about 10 CCs per kilo body weight. So 500 to 700
25 CCs.

1 Q. 500 CCs for a man, a grown man that's my size. 500 CCs.
2 This is a water bottle, that's 500 CCs of water.
3 500 milliliters of water is the same volume of water as CCs of
4 air.

5 A. That's more like 250 to me. Is that what it says? Does
6 it say 500?

7 Q. It says 500. So you would agree with me that one breath
8 is 500 cubic centimeters of air, right?

9 A. Yes.

10 Q. Okay. And that if you breathe 16 breaths a minute and
11 there's 500 cubic centimeters of air for each breath and
12 you're in an environment where there is one asbestos fiber per
13 cubic centimeter, that means you're breathing 8,000 fibers in
14 a minute, right?

15 A. Seems right.

16 Q. Okay. And just so we get on the record what a cubic
17 centimeter is, a cubic centimeter is about the size of a sugar
18 cube. It's a centimeter by a centimeter by a centimeter,
19 right?

20 A. That's the example I was going to give.

21 Q. Okay. And so if it's 8,000 fibers in a minute, it's
22 120,000 fibers in 15 minutes at one fiber CC, right? Third
23 grade math. Agree with that?

24 A. Yes.

25 Q. And it's 480,000 fibers in an hour, right?

1 A. Correct.

2 Q. And it's a little less than 4 million fibers of asbestos
3 breathed in one working day if you're in an environment with
4 one fiber per cubic centimeter, correct?

5 A. Right.

6 Q. Now, are you familiar that there are various estimates of
7 the exposure -- of the amount of asbestos found in the
8 background ambient air, correct?

9 A. Correct.

10 Q. I'm going to put up one from the ATSDR in 2001 in just a
11 second which is a little bit lower than what you have in your
12 textbook, but you've got a page in your asbestos-associated
13 diseases book that has a table that summarizes asbestos
14 exposure samples in different environments, right?

15 A. Yes.

16 Q. And you know, I'll show it to you in a minute, but I'm
17 going to do this with the ATSDR because it makes the math
18 easier, frankly. While I can do math quick in my head, I
19 sometimes -- anybody can make a mistake without a calculator,
20 right? You would agree that anybody can make a mistake with
21 math sometimes.

22 A. Well, I sure have.

23 Q. Huh?

24 A. I sure have.

25 Q. So just because you're not perfect with math doesn't mean

1 that your conclusions aren't valid, correct?

2 A. Right.

3 Q. So in your book, the median fiber per CC concentration
4 for the air of 48 United States cities is .00005. Four zeros
5 and a five, is what it says in your book at page 26. Right?

6 A. I'd have to see it.

7 MR. FINCH: Can I approach the witness, Your Honor?

8 A. Yes, that's the concentration of fibers per CC, yes.

9 Q. And it's -- and it's what I just said it was which is
10 zero point four zeros and then a five, right?

11 A. Right.

12 Q. Now, you're aware that the ATSDR, which is the Agency for
13 Toxic Substances and Disease Registry, they published a big,
14 fat book on asbestos and disease in about 2001, right?

15 A. Yeah.

16 Q. And they stated that since there are 1 million cubic
17 centimeters in a cubic meter, there typically would be 0.00001
18 fibers per milliliter of asbestos in air in rural areas.
19 Typical levels found in cities are about 10-fold higher.

20 And I'm going to start with their number for rural areas
21 and then I'm going to use your number out of your book for the
22 cities just so the math is easy.

23 If somebody is resting, they're going to be breathing a
24 little bit less maybe than if they are working, right? Little
25 bit less frequently. Would you agree with that?

1 A. Yeah, the minute ventilation if they are resting would be
2 less than if they weren't.

3 Q. Okay. So 12 breaths a minute if you are just doing
4 nothing is a reasonable estimate of how many times you breathe
5 a minute, right?

6 A. Yes.

7 Q. And as we established, one breath is 500 CCs of air.
8 That may be a little bit on the low side for a grown man, but
9 that's a reasonable estimate of air per breath, right?

10 A. Yes.

11 Q. And again, 12 breaths at 500 CC of air times 6,000 CCs in
12 a minute, that's 6,000 cubic centimeters of air in a minute.
13 You would agree with that, right?

14 There's 360,000 cubic centimeters of air in an hour.
15 That's just multiplying 6,000 by 60. That gets you to
16 360,000, right?

17 A. Yes.

18 Q. And if you do that, since you've got to breathe 24 hours
19 a day or you're going to die, that gets you to 8,640,000 cubic
20 centimeters of air breathed in a day. That's a reasonable
21 estimate of how much cubic centimeters of air someone breathes
22 in a day, right?

23 A. Seems reasonable.

24 Q. Okay. And if the fiber concentration in the air, the
25 asbestos fiber concentration is .00001. If you do that math,

1 you get 86 fibers in a day at rural ambient air concentration,
2 correct?

3 A. Yes.

4 Q. All right. So -- and this is the page out of your book I
5 just showed you, right, with the air of 48 U.S. cities, median
6 concentration is .00005. See that, Dr. Sporn?

7 A. Yes.

8 Q. Okay. So if in the rural air, if you breathe in 86
9 fibers a day, in the cities' ambient asbestos exposure, all
10 you have to do to figure out what the difference is between
11 the rural and cities is multiply by 5, right?

12 A. Owing to the higher concentration.

13 Q. In the cities.

14 A. Right.

15 Q. So ambient air exposure, whatever people are breathing --
16 and these are studies based on what the ambient air exposure
17 was 10, 20 years ago, correct?

18 A. Yes.

19 Q. And you would agree with me that as we have used less
20 asbestos in the world or in the United States, then you would
21 expect the ambient air concentrations to go down over time?

22 A. I don't know if that's necessarily true because most --
23 because we're still finding commercial amphiboles in people's
24 lungs and those have been out of the marketplace for a number
25 of years. So I don't know.

1 Q. Okay. All right. So let's just say that it's the same
2 now as it was ten years ago. The ambient air concentration is
3 432 asbestos fibers in a day if you are in a city, right?

4 That's a reasonable estimate.

5 A. Seems to be based on your math.

6 Q. As compared to 3.8 million fibers in one working day at
7 one fiber per CC, correct?

8 A. At one fiber per CC -- yeah.

9 Q. Okay. And at 0.1 fibers per CC, and I actually didn't do
10 the math right here because if you divide -- all you have to
11 do to figure out how many fibers you breathe at the current
12 OSHA permissible exposure limit of 0.1 fiber per CC is to
13 divide that 3.8 million figure by 10, right?

14 A. Right.

15 Q. And so one day of working in an environment with 0.1
16 fibers per cubic centimeter of asbestos is 364,000 -- or
17 384,000 a day versus 432 fibers in ambient air, right?

18 A. Yes.

19 Q. All right. And so you could get to in a couple of weeks
20 working at 0.1 fiber per CC asbestos, you could get to as many
21 fibers as you breathe in ambient air in your whole life,
22 right? If you did the basic math.

23 A. Again, that would depend on what size fibers they were
24 and what length they were, where they were deposited. If they
25 were -- if they were less than half -- less than 5 microns, if

1 they were long, curly fibers greater than 30 -- it's hard --
2 it's hard to know with those raw numbers how many of those
3 asbestos fibers will be deposited in the parts of the lung
4 that are involved in gas exchange.

5 Q. I'm just asking about what comes into your lung. And
6 you --

7 A. No --

8 Q. And the way historically they measure asbestos fiber in
9 the air, you would only measure fibers longer than 5 microns
10 for fibers in the air, right? Do you agree with that?
11 Historically they're only measuring fibers longer than 5
12 microns.

13 A. Okay.

14 Q. Now, you're familiar with that document, the Helsinki
15 criteria?

16 A. Sure.

17 Q. Your colleague Victor Roggli was a contributor to it,
18 correct?

19 A. Sure.

20 Q. That was a document, it was a group of --
21 multidisciplinary gathering of pathologists, radiologists,
22 occupational and pulmonary physicians, epidemiologists,
23 toxicologists, industrial hygienists, and clinical and
24 laboratory scientists specializing in tissue fiber analysis.
25 Collectively, the group has published over a thousand articles

1 on asbestos and asbestos-associated diseases. That article
2 was published in 1997, correct?

3 A. Correct.

4 Q. And what they state in the first page is, "In general,
5 reliable work histories provide the most practical and useful
6 measure of occupational asbestos exposure."

7 Dr. Roggli agreed with that in 1997, correct?

8 A. That's what it says in Helsinki. But again, it's a big
9 document and it is -- and it is not one that I don't believe
10 from my perusal of it to speak to causation of fiber type.

11 Q. Well, doesn't the document actually say all types of
12 malignant mesothelioma can be induced by asbestos, with the
13 amphiboles showing greater carcinogenic potency than
14 chrysotile?

15 A. Well, again, I would agree with that statement with the
16 caveat that, again, you have to -- when you're talking about
17 chrysotile, you cannot frame any type of discussion about
18 chrysotile without breaking it down. Is it chrysotile fibers
19 you're dealing with? Is it chrysotile dust you're dealing
20 with? Is it chrysotile ore? Is it chrysotile dust from
21 mines?

22 Q. This document was published in the medical literature and
23 it states that "an occupational history of brief or low level
24 exposure should be considered sufficient for mesothelioma to
25 be designated occupationally related to asbestos exposure.

1 "A history of significant occupational, domestic or
2 environmental exposure to asbestos will suffice for
3 attribution."

4 That's what the Helsinkey criteria says, correct?

5 A. It does. But I would submit to you that a -- someone who
6 is, let's say for argument purposes, a shade-tree mechanic,
7 that those -- the people who penned the Helsinki criteria
8 would not look upon that as significant occupational exposure.

9 Q. And there is no qualification in those statements about,
10 oh, this doesn't count for chrysotile, this doesn't count for
11 brakes, correct?

12 A. No.

13 Q. All right. Now, you talked a lot about your work with
14 Dr. Roggli, and you and Dr. Roggli have worked together for
15 what, almost 20 years?

16 A. Yes.

17 Q. You know that Dr. Roggli has testified on multiple
18 occasions in asbestos litigation, correct?

19 A. Yes.

20 Q. Let me show you a sworn statement that Dr. Roggli
21 testified to.

22 Dr. Roggli lives in the county of Durham, right?

23 A. He does. Been to his house.

24 Q. "My name is Victor Roggli. I am over the age of 18 years
25 and fully competent to make this affidavit. The facts stated

1 in this affidavit are within my personal knowledge and are
2 true and correct."

3 You wouldn't expect Dr. Roggli to lie about something,
4 would you?

5 A. No, I wouldn't expect him to lie about anything.

6 Q. "While there is no question among experts in the field
7 that chrysotile dust causes the disease mesothelioma, there is
8 some dispute regarding whether it is the chrysotile itself or
9 the tremolite contaminant of the chrysotile dust or both that
10 causes the mesothelioma. The resolution of that dispute is,
11 however, purely an academic exercise. Over 99 percent of the
12 chrysotile used in the United States was mined in Canada.
13 Every published study addressing the issue indicates that
14 there is some degree of tremolite contamination in all
15 chrysotile ore mined in Canada. Further, there is no evidence
16 that the tremolite contamination is removed during the
17 processing of the chrysotile ore."

18 That's what Dr. Roggli swore to in this affidavit,
19 correct?

20 A. Can you tell me when the affidavit was.

21 Q. The affidavit was in a case called Innerarity and it was
22 in 2001. Let me just see if you agree that Dr. Roggli's
23 opinions in 2001 are consistent with the affidavit.

24 "The scientific and medical community has yet to
25 determine the level of exposure to asbestos below which

1 mesothelioma will not occur. While there is no threshold,
2 there is insufficient evidence to implicate levels of asbestos
3 exposure, exposure to asbestos that occur as a result of
4 background or ambient air exposure. Very low levels of
5 exposure above background, however, have been demonstrated to
6 cause mesothelioma.

7 "It is also my opinion that it is the total dose of
8 asbestos, regardless of fiber type, that the patient
9 experiences that causes the disease.

10 It is further my opinion that each and every exposure to
11 asbestos that an individual with mesothelioma experienced in
12 excess of a background level is a substantial contributing
13 factor in the development of the disease."

14 I read that correctly, correct?

15 A. Yes.

16 Q. And substantial contributing factor, that's not a concept
17 that you and Dr. Roggli or any doctor talks about in the real
18 world of seeing patients. That's a question you get asked in
19 front of a jury in the context of a legal case, correct? It's
20 a mixed question of medical opinion, but doctors don't go
21 around saying in my opinion smoking was a substantial
22 contributing factor in causing this man's lung cancer. They
23 say his lung cancer was caused by smoking, right?

24 A. Ordinarily when I issue a diagnosis of mesothelioma, I
25 don't opine as to the cause one way or another unless I'm

1 specifically asked to.

2 Q. And here Dr. Roggli was specifically asked and he was
3 specifically asked in the context of a case involving Boyce
4 Innerarity, "I have reviewed Boyce" -- I can't pronounce the
5 gentleman's name -- "Innerarity's deposition testimony
6 regarding his work history. Based on his testimony, it is my
7 opinion that his exposure to insulation materials containing
8 asbestos was not the sole cause of his disease. He was
9 exposed to many different products that contained asbestos.
10 Specifically, I reviewed Mr. Innerarity's testimony regarding
11 the manner in which he removed Garlock asbestos-containing
12 gaskets, and to the extent that on repeated occasions over a
13 career spanning three decades Mr. Innerarity as a regular part
14 of his job removed dry, adhered Garlock gaskets containing up
15 to 85 percent chrysotile asbestos from pipe flanges using
16 electric wire brushes and wore no respiratory protection, and
17 to the extent that such activity created dust levels
18 substantially above background, it is my opinion that exposure
19 to dust from the Garlock gaskets was a substantial
20 contributing cause in the development of his mesothelioma."

21 That's what Dr. Roggli wrote in an affidavit he signed in
22 2001, correct?

23 A. Yes. And was there a question to me there or just that
24 Dr. Roggli wrote that affidavit?

25 Q. Yeah, Dr. Roggli wrote that affidavit in 2001.

1 A. He wrote that affidavit, yeah.

2 Q. Okay. Now, you talked about Dr. Hammar on direct,
3 correct?

4 A. Yes.

5 Q. He was a mentor of yours when you were out in Washington
6 state, correct?

7 A. Yes.

8 Q. And you know that Dr. Hammar has published numerous
9 articles in the peer reviewed literature on the causation and
10 diagnosis of asbestos-related diseases, correct?

11 A. Yes.

12 Q. This textbook, "Dail and Hammar's Pulmonary Pathology" is
13 a well-regarded pathology textbook, correct?

14 A. Yes. I wrote the chapter on occupational lung disease in
15 it.

16 Q. Okay. There's also a chapter that Dr. Hammar wrote
17 entitled "Neoplasms of the Pleura." You know that that
18 chapter is in the book, correct?

19 A. Yes.

20 Q. Okay. And what Dr. Hammar writes is "No lower (minimum)
21 threshold level of exposure to asbestos has been delineated
22 below which there is no increase in the risk of malignant
23 mesothelioma. And most authorities approach causation of
24 mesothelioma by asbestos from the perspective of a no
25 threshold model. One factor that emerges from the Peto model

1 and its modifications is that where there are multiple
2 asbestos exposures, each contributes to cumulative exposure
3 and hence to the risk and causation of malignant mesothelioma
4 within an appropriate latency interval."

5 That's what Dr. Hammar wrote in his pathology textbook
6 that was published in 2008 and is on the library shelves of
7 many practicing pathologists around the country, correct?

8 A. Yes.

9 Q. Finally --

10 A. And am I just supposed to agree that that's out there or
11 was there a question?

12 Q. You agree that that's out there, correct?

13 A. I agree that that's out there.

14 Q. Okay. And you agree that Dr. Hammar is a well-respected,
15 well-regarded scientist in the field of asbestos and disease
16 causation.

17 A. I would agree with that. He's also one that both
18 Dr. Roggli and I find myself in significant and substantial
19 disagreement on any number of cases, especially in the area of
20 litigation.

21 Q. Okay. Let's talk about something I think -- this is my
22 last topic and I don't think you're going to disagree with
23 anything I say or anything any other doctors say.

24 Mesothelioma is almost invariably a fatal disease,
25 correct?

1 A. Unfortunately, yes.

2 Q. And what often happens is -- one of the first things that
3 happens is people get diagnosed with mesothelioma or they
4 think they might have mesothelioma and there will be a pleural
5 effusion or some other kind of change that's causing them
6 pain, right?

7 A. The pleural effusion might cause shortness of breath. It
8 rarely causes pain. But they usually present with pains
9 referable to the mesothelioma in their chest.

10 Q. And oftentimes they have fluid drained from their body.
11 I've seen -- I've had client after client in the medical
12 records, two liters of fluid taken out of the body. That's
13 one of the -- that's oftentimes a consequence of having
14 mesothelioma, correct?

15 A. Yes. And I personally have drained over liters and
16 liters and liters and liters of fluid off the chest of people
17 with mesothelioma.

18 Q. And then as mesothelioma progresses, it gets -- it causes
19 a constricting of people's ability to breathe in many cases,
20 correct?

21 A. Yeah. It causes breathing impairments due to reduction
22 of lung volume, yes.

23 Q. And it causes people to undergo severe debilitating pain
24 in many cases, right?

25 A. It's a terrible disease.

1 Q. It's a terrible disease. And the survival rate for
2 people with pleural mesothelioma, most of them are dead within
3 a year or two, right?

4 A. Fortunately that's changing, but overall historically the
5 prognosis is measured in months to a few years.

6 Q. And in some people, if they're really young and in good
7 shape, they'll try desperately to stay alive by having one of
8 their lungs removed in a procedure called an extrapleural
9 pneumonectomy where they take out the lung where the pleura is
10 that has the mesothelioma in an attempt to extend their life,
11 correct?

12 A. Yes. And to reduce pain, yes.

13 Q. And that's a procedure that you know that's done in New
14 York, it's done in Philadelphia, it's done by Dr. Sugarbaker
15 in Boston, correct?

16 A. And probably most of all at my hospital.

17 Q. And at Duke. I wasn't meaning to slight Duke.

18 And most of the time people with mesothelioma, even if
19 they have that surgery, the tumor comes back and they die from
20 it, correct?

21 A. Unfortunately.

22 Q. And they die in tremendous pain, correct? Many, many
23 people with mesothelioma.

24 A. It's a terrible disease.

25 MR. FINCH: That's all I have, Your Honor.

1 THE COURT: Anything else, Mr. Schachter?

2 MR. GUY: Your Honor...

3 THE COURT: I'm sorry. Mr. Guy. I forgot about
4 you.

5 MR. GUY: That's okay. As long as you don't forget
6 about me completely.

7 CROSS EXAMINATION

8 BY MR. GUY:

9 Q. Dr. Sporn, my name is Jonathan Guy. I represent the FCR
10 in this case.

11 A. Good morning -- or afternoon.

12 Q. You wrote your article about malignant mesothelioma and
13 occupational exposure to asbestos in 2002, correct?

14 A. Yes.

15 Q. And was that published so it was available to the public?

16 A. Yes.

17 Q. Would it have been available to asbestos defendants like
18 Garlock?

19 A. Sure.

20 Q. And your book, when did you first write your book?

21 A. That was published in 2004.

22 Q. And that's also obviously available to the public?

23 A. You can get it on Amazon.

24 Q. Is there any reason why Garlock wouldn't be able to
25 consider the position that you're taking and have taken

1 concerning chrysotile or mesothelioma in evaluating whether to
2 litigate and settle cases?

3 A. That is a decision that law firms and/or commercial firms
4 make and I have no opinion in that regard.

5 Q. When you first wrote your article, you were at Duke,
6 correct?

7 A. Yes.

8 Q. And you could have been reached by Garlock over the
9 telephone quite easily, correct?

10 A. By telephone I'm hard to reach. Maybe by email.

11 MR. GUY: No further questions, Your Honor.

12 THE COURT: Mr. Schachter.

13 REDIRECT EXAMINATION

14 BY MR. SCHACHTER:

15 Q. Sir, you were asked some questions about various agencies
16 that have looked at the mining studies that you told us about
17 on direct, and looked at other studies and opined that
18 chrysotile can cause mesothelioma based on those situations.

19 Is that inconsistent with your opinion as long as we
20 understand what chrysotile --

21 A. Yes. I think that -- whenever I enter into a discussion
22 regarding the potential for chrysotile to cause mesothelioma,
23 I want to make sure that the parties to the discussion, that
24 we're all on the same footing on what we're talking about.

25 Are we talking about chrysotile that's in the dust of mines

1 and chrysotile producing areas in Quebec? Are we talking
2 about the chrysotile that was used historically in friction
3 products? What are we talking about? And we need to be very,
4 very specific about it in our discussion in that regard.

5 Q. Now, on your opinion on -- you were asked some questions
6 about no safe level and I don't think you were able to fully
7 explain. Has science yet established at what level the danger
8 of asbestos related mesothelioma rises, what level of exposure
9 creates the danger?

10 A. Not for the amphibole forms of asbestos. But if you --
11 if you look to what has been published in the literature
12 regarding -- regarding chrysotile -- and this is chrysotile
13 dust, dust from mines, or dust that has been liberated in
14 areas where there are a lot of mines such as in Thetford,
15 Quebec, those -- again, there is no, quote, safe level, but
16 there's been a range of levels that have been observed and
17 those are in the hundreds of fibers -- of fiber years.

18 Q. And if for regulatory purposes an entity wants to assume
19 equal potency, does that mean that for a scientific fact the
20 fibers on a fiber per fiber basis are equally potent?

21 A. No. For a public health model, they would take things --
22 I would assume like they do in other models, take things back
23 to zero. The risk begins with that very first fiber. That's
24 a separate argument from a scientific concern.

25 Q. Now, you're acquainted with Dr. Roggli, of course, right?

1 A. Yes.

2 Q. And you were shown an affidavit from 12 years ago or 13
3 years ago purportedly in a case.

4 A. Yes.

5 Q. Has he been testifying to the fact that low dose products
6 like chrysotile based on the current science are not a cause
7 of mesothelioma?

8 A. Yes, he has.

9 Q. All right. And when it comes to gaskets, you were also
10 shown -- you were also -- they talked to you a lot about
11 Mr. Nicholson or Dr. Nicholson.

12 Can you put up the exhibit.

13 You're familiar with William Nicholson. And he was
14 discussed in -- he was about -- he was asked about whether
15 gasketting work created a risk of mesothelioma, and prepared
16 an affidavit opining that he "would conclude that the relative
17 contribution of gasketting work to cancer, including
18 mesothelioma, originating from workplace exposures is
19 minuscule. Furthermore, the contribution to any non-malignant
20 asbestotic disablement is literally nonexistent. From the
21 data that exists from clinical findings of asbestosis exposed
22 workers, it is clear that the exposure from installation or
23 removal of gasket materials would not lead to any discernable
24 disablement among those undertaking such work or among those
25 exposed indirectly."

1 That was Dr. Nicholson in 1993 -- 83.

2 Is there anything inconsistent in what Dr. Nicholson was
3 saying with the opinions that you've expressed here today?

4 A. No, sir.

5 Q. And based upon the current science, is our gaskets a
6 cause of mesothelioma in human beings?

7 A. No, sir.

8 MR. SCHACHTER: Thank you.

9 THE COURT: You can step down. Thank you, Dr.
10 Sporn.

11 THE WITNESS: Thank you, Your Honor.

12 (Witness stepped down.)

13 THE COURT: Do you have another witness you want to
14 start today?

15 MR. SCHACHTER: Mr. Harris may.

16 MR. HARRIS: Yes, Your Honor.

17 THE COURT: Okay.

18 MR. HARRIS: At this time we call -- Garlock calls
19 Larry Liukonen.

20 LARRY RAY LIUKONEN,
21 being first duly sworn, was examined and testified as follows:

22 DIRECT EXAMINATION

23 BY MR. HARRIS:

24 Q. Please tell us your name.

25 A. My name is Larry Ray Liukonen.

1 Q. Where are you from?

2 A. I'm from Burleson, Texas.

3 Q. What do you do for work?

4 A. I'm an industrial hygiene consultant.

5 Q. Are you a certified industrial hygienist?

6 A. Yes, I am. I have been since 1976.

7 Q. This case involves individuals who allege worked with
8 asbestos gaskets and packings. Have you studied potential
9 exposures associated with that work?

10 A. Particularly I've studied exposure with work with
11 gaskets.

12 Q. We've heard about a study on gaskets by the United States
13 Navy in 1978. Are you familiar with it?

14 A. Yes, I am.

15 Q. How so?

16 A. I was the lead author.

17 Q. What was the purpose of your study?

18 A. In 1978 the navy at least knew -- we didn't always do it,
19 but at least we knew what we should do with friable thermal
20 insulation products. So someone was wondering if they should
21 look at other more traditionally non-dusty products and see if
22 any modifications needed to be made to work procedures.

23 So they asked all of the industrial hygienists in the
24 navy if anyone had looked at it and everyone replied, no, we
25 really haven't taken a look at it. So they asked us in

1 Bremerton to design a study to evaluate all aspects of the
2 life cycle of a gasket.

3 Q. When you were with the U.S. Navy, did you also research
4 and assess exposures to insulation used on ships?

5 A. Yes, I did.

6 Q. Have you brought with you documents from your work in the
7 navy in the 1970s and the work of others that you have relied
8 upon?

9 A. Yes.

10 Q. And have you prepared some slides to help us understand
11 what the potential exposures were historically from working
12 with asbestos gaskets and packing and insulation products?

13 A. Yes, I have.

14 Q. Before I ask you about that research, I would like to ask
15 you a few questions about what qualifies you to discuss these
16 matters. What was your educational background?

17 A. I have a bachelor of science in the biological sciences
18 from the University of Minnesota. After that I went directly
19 to graduate school and I received a master of science in
20 environmental health where I studied air pollution and
21 industrial hygiene.

22 Q. At this time it would probably be a good idea if you told
23 us what industrial hygiene was.

24 A. Sure. Basically, what we do is try and prevent
25 occupational diseases. We spend a lot of time in the

1 workplace looking at what people do, what chemicals or
2 materials they work with, what sort of work practices they
3 use. We -- if necessary we make measurements or we collect
4 air samples or possibly noise measurements. We compare those
5 results to recommended standards; and if necessary, we
6 recommend corrective action.

7 Q. In your graduate studies, did you study asbestos or
8 potential asbestos exposures in the workplace?

9 A. Yes.

10 Q. After you received your degree, where did you go to work?

11 A. After my -- I received my master's degree, I accepted a
12 commission in the United States Navy and I did industrial
13 hygiene for them on active duty for three years.

14 Q. All right. It indicates here from 1972 to 75 you were
15 with the U.S., is it naval reserve?

16 A. I was in the naval reserve, active duty and naval
17 reserve.

18 Q. Why did you join the navy?

19 A. When I went to -- when I was in my graduate program,
20 several of my co-students were being sent there by the
21 military. Military was sending people back to get their
22 master's degree in industrial hygiene. Military was actively
23 looking for industrial hygienists, so I thought that was a
24 good opportunity.

25 Q. So you were actually in the navy. Did you have a rank in

1 the navy?

2 A. Yes, I did. I started out as lieutenant junior grade,
3 lieutenant JG, and I finished as a lieutenant commander.

4 Q. It says here you were in Cincinnati; is that correct?

5 A. That's correct.

6 Q. What was the nature of that work?

7 A. We were basically a consulting group. We had
8 occupational health physicians, we had occupational nurses and
9 industrial hygienists. And we provided assistance to naval
10 facilities primarily in the U.S., but some worldwide. When
11 they needed special assistance for a project, either they
12 didn't have the expertise themselves or maybe they didn't have
13 enough staff, then we would go and assist with those projects.

14 Q. And you did that for three years; is that correct?

15 A. I did, yes.

16 Q. And then you left the navy; is that correct?

17 A. I left active duty.

18 Q. While you were there from '72 to '75, did you have any
19 contact with asbestos products or asbestos that's used in the
20 navy?

21 A. Not very much. The only one that I specifically recall
22 is I was sent to a shipyard that was having a little problem
23 with a ventilation system that they were using for asbestos
24 fabrication. They needed somebody to come and take a look at
25 it. And two of us went down there and helped with that

1 project.

2 Q. It looks like in 1975 you then left and went to Naval
3 Regional Medical Center at Bremerton, Washington. Now, were
4 you in the navy at this point?

5 A. No, then I was a civilian.

6 Q. So why did you go to Bremerton? You're out of the navy
7 now, why did you decide to go back and work for the navy as a
8 civilian?

9 A. The -- because the -- the really good industrial hygiene
10 jobs in my opinion were at the shipyards and so I wanted to
11 go -- number one, I wanted to go to one of the places that had
12 a shipyard. And I picked Bremerton as my first choice because
13 they had a reputation of having the best industrial hygiene
14 program in the navy. So that's where I wanted to go.

15 Q. In 1976 we saw on the prior slide that you became a
16 certified industrial hygienist.

17 A. That's correct.

18 Q. Why did it take you so long to become a certified
19 industrial hygienist?

20 A. They require that you have five years of professional
21 experience before you can take the examination. When you have
22 an advanced degree, you can knock off one year, which is what
23 I did.

24 Q. When you got to Bremerton, what were the industrial
25 hygiene concerns there?

1 A. We had a lot of concerns because we had a ten thousand
2 person shipyard. We covered the whole Pacific Northwest. We
3 had air stations and ammunition facilities and lots of things.
4 Our main concerns were asbestos from friable thermal
5 insulation. We had concerns with heavy metals like cadmium
6 and lead. We had concerns with noise. We had concerns with
7 solvents.

8 Q. And what were the asbestos concerns specifically?

9 A. That would be rip out -- removal of thermal insulation
10 aboard ship when ships came in for overhaul.

11 Q. You mentioned rip out. Tell us what rip out is.

12 A. Rip out is removal. It's the thermal insulation and --
13 when a ship comes in for overhaul, there's a lot of that
14 that's removed and it can be very dusty. And it was something
15 we were very concerned about.

16 Q. Had the asbestos controls been implemented at Puget Sound
17 Naval Shipyard when you arrived or before you arrived in 1975?

18 A. There were some controls that were in place. Not very --
19 pretty primitive by today's standards. And we had a difficult
20 time enforcing even the minimal controls that we did have. We
21 did not get a lot of cooperation from some of the supervisors;
22 therefore, not a lot of cooperation from some of the workers.

23 Q. When you say minimal controls, what type of controls are
24 we talking about?

25 A. Well, theoretically they were to wet the material when

1 they removed it. Often didn't happen.

2 In terms of keeping people out of the area, it was simply
3 a rope and a sign.

4 And you could work within 5 feet of someone removing
5 thermal insulation without any protective equipment
6 whatsoever.

7 Q. A rope. What about plastic sheeting and creating a tent
8 or enclosure, was that going on in 1975 in Puget Sound?

9 A. It was not.

10 Q. Did you conduct any air monitoring during the thermal
11 insulation removal while you were there?

12 A. Yes.

13 Q. What type of exposures did you find even during this
14 period of minimal controls?

15 A. Our goal, if everybody did everything perfectly, our goal
16 was to stable .02 fibers per CC. That usually didn't happen,
17 and we measured 5, 10, 20 commonly and levels over a hundred
18 and even over 200 fibers per CC.

19 Q. Okay. To put that in perspective, the OSHA permissible
20 exposure limits in 1975 would have been the original one of
21 five fibers per CC?

22 A. That's correct.

23 Q. Now, were there any controls in place when you arrived at
24 Bremerton with respect to doing any work with gaskets and
25 packing?

1 A. No, there were no controls whatsoever for gaskets or
2 packing.

3 Q. And how long did you stay with the navy as a civilian
4 industrial hygienist?

5 A. Four years.

6 Q. That takes us to 1979, I believe. And then from there
7 you worked for the railroad for almost ten years or eight
8 years.

9 A. Eight and a half years.

10 Q. What type of work did you do there?

11 A. I was the first industrial hygienist at that company and
12 did a lot of noise monitoring, sampling for diesel exhaust,
13 again, heavy metals, silica. Asbestos was a pretty minor
14 concern there.

15 Q. And then in, I believe it was 1987, you formed a company
16 with a partner, and then in '88 you formed your current
17 company; is that correct?

18 A. That's correct.

19 Q. And that's Technical Safety and Health Consulting, Inc.

20 A. That's correct.

21 Q. And what type of work have you done since then?

22 A. We do comprehensive industrial hygiene. We've
23 remained -- done a lot of it in the railroad industry. Do a
24 lot of work for the railroad industry. A lot of routine air
25 sampling and noise monitoring. I've been involved in

1 litigation-related activities the entire time, more so towards
2 the end than I was at the beginning.

3 MR. HARRIS: At this time, Your Honor, we tender
4 Mr. Liukonen as an expert in industrial hygiene.

5 MR. GEORGE: No objection, Your Honor.

6 THE COURT: All right. He'll be admitted as such.

7 Q. Mr. Liukonen, you've also published in the peer reviewed
8 literature; is that correct?

9 A. That's correct.

10 Q. It looks like we've got two articles here. Can you tell
11 us what those were.

12 A. Yes. The first one is an article -- a study that we did
13 on exposure of railroad train crews to particulates from
14 diesel engines, such as locomotives is what we were looking
15 at.

16 The second one is a study I did with Dr. Weir which was
17 regarding asbestos exposure from working with a diesel engine,
18 removing the gasket material from a diesel engine.

19 Q. What type of gasket material? Were these special engine
20 gaskets?

21 A. Well, they're basically the same type of gaskets you'd
22 find everywhere else. There was varying percentages of
23 asbestos. I think like some were as low as 5 percent. Others
24 were probably as high as 75 or 80 percent asbestos.

25 Q. Were they compressed asbestos sheet gaskets?

1 A. Yes, they were.

2 Q. I would like to ask you now about the study that you did
3 for the navy. This was in 1978; is that correct?

4 A. That's correct.

5 Q. There you are as L.R. Liukonen as the first author.

6 A. That's correct.

7 Q. The second author is Kenneth Still. And you understand
8 that he's been designated as an expert witness in this case by
9 Garlock; is that correct?

10 A. Yes.

11 Q. And then the third author is R.R. Beckett, and you
12 understand that he has been designated as an expert witness by
13 the committee; is that correct?

14 A. Yes, I do.

15 Q. You've read his report?

16 A. Yes, I have.

17 Q. And you've read his deposition; is that correct?

18 A. Yes.

19 Q. I believe you told us generally how this study came
20 about. Could you tell -- remind me again exactly why you did
21 this study.

22 A. The navy wanted to know at this point whether they needed
23 to modify any procedures to keep exposures below the PDL at
24 the time. And also there was -- the navy had a medical
25 monitoring program that begin at .1 fibers per CC. So another

1 goal of the study was to determine if people that worked with
2 asbestos gaskets needed to be enrolled in this medical
3 monitoring program.

4 Q. Was the .1 fibers per CC an 8-hour time-weighted average?

5 A. It really was, but as industrial hygienists generally, we
6 tend to be very conservative and tend to think that -- we tend
7 to -- we tend to think in terms of the exposures for
8 short-term even though it really is a time-weighted average.

9 Q. And at this point in time, the 8-hour time-weighted
10 average in 1978 had been lowered to two fibers per CC; is that
11 right?

12 A. That's correct.

13 Q. Had you studied potential exposures to
14 asbestos-containing gaskets or packing in the shipyard before
15 this study?

16 A. We had two samples that we had collected before this
17 time.

18 Q. And what were those samples in relation to?

19 A. They were in relation to an operation where -- gaskets
20 are actually manufactured in the shipyard. There's a large
21 number of gaskets that would be used in the shipyard, so
22 instead of everyone making their own, we had a couple of
23 employees that that was their job was to stamp out gaskets.
24 One of them used an automatic punch press. The other used a
25 mallet with a punch to punch the bolt holes in the gaskets.

1 And that's where those two samples had been collected.

2 Q. There were only two workers that were doing that type of
3 work?

4 A. That's correct.

5 Q. And how many other people would have been working or
6 coming in contact with asbestos gaskets during this time in
7 the shipyard?

8 A. Oh, probably thousands of other people would use
9 asbestos-containing gaskets.

10 Q. The -- what type of exposures did you find for this
11 manufacturing type operation?

12 A. When we did those two samples in 1975, we included
13 this -- those in this study as well. We reported three fibers
14 per CC for one sample and five fibers per CC for the other
15 one.

16 Q. When you received those samples, did you take any action?

17 A. We did. We recommended that they be included in the
18 medical monitoring program and we recommended that that area
19 be thoroughly cleaned.

20 Q. Why hadn't a report been done on gasket operations before
21 your study?

22 A. Well, as an industrial hygienist I didn't really consider
23 it as a real potential for exposure. Gaskets are encapsulated
24 materials. They're not dusty materials. They're
25 insignificant compared to the exposures that we would expect

1 from friable thermal insulation.

2 Q. Was sampling done under actual real world type
3 conditions?

4 A. Absolutely. We collected them under normal work
5 conditions in shops and aboard ship.

6 Q. Did the study have anything to do with litigation at the
7 time?

8 A. No, none whatsoever.

9 Q. What operations did you study?

10 A. We studied all aspects. We tried to determine all
11 aspects of the life cycle of a gasket.

12 Let me -- there we go.

13 We looked at storage, when the gasket materials are
14 received and stored for use. We looked at the hand gasket
15 punching; hand operated mechanical punching, which was
16 basically pulling a lever down to punch a hole in the gasket;
17 machine punching; hand shaping; machine sheering; machine
18 nibbling; installation; removal with concurrent installation;
19 removal and hand scraping; removal and wire brushing; and then
20 clean up following removal. By clean up following removal, I
21 don't mean cleaning the floor or something like that at the
22 work area. I mean cleaning up the flange, the gasket remnants
23 that are left on the flange.

24 Q. After the gasket is removed?

25 A. Yes.

1 Q. And that was a separate sampling that was done; is that
2 right?

3 A. That's correct.

4 Q. And so these are -- are these all the operations
5 involving gaskets in the shipyards that you could -- the
6 shipyard that you could identify?

7 A. Yes.

8 Q. Which of these operations would be typical for someone
9 that is working with gaskets, like a pipefitter or a machinist
10 make -- a pipefitter in the shipyard or a machinist made in
11 the navy?

12 A. They would typically be working with the -- here they're
13 broken down. Would be the ones on the left side. This is the
14 ones that most people would be working with. The ones that we
15 consider as secondary manufacturer. Primary manufacturer to
16 me is someone that actually makes the gasket sheet. Secondary
17 manufacturer is where they stamp the gasket out of that sheet.

18 Those are the processes on the right-hand side. There
19 was a limited number of people in the shipyard that did that
20 work, was probably two people full-time and another person
21 part-time. Whereas the left side, that's the end user and
22 that's -- we have large numbers of people to do those sorts of
23 activities.

24 Q. So a company like Garlock would manufacture compressed
25 asbestos sheet. It would get sold to or distributed to the

1 shipyard and then people -- at least two workers, I guess, in
2 the shipyard would then cut from the sheets, cut out the
3 gaskets that would then be used on the ships in the shipyard;
4 is that correct?

5 A. That's correct.

6 Q. Okay. And you only had a couple of people there at the
7 Bremerton shipyard doing that type of work; but on the
8 left-hand side, the end user activities, how many workers were
9 we talking about?

10 A. I would suspect thousands.

11 Q. Okay. So I want to focus mostly on those people for the
12 purposes of this exam.

13 We've talked about fibers per CC in terms of exposure
14 limits and so forth. Can you explain a little bit more about
15 that process. How are the -- how are air samples collected?

16 A. I brought an air sampling pump here that we can
17 demonstrate. We use -- it's a battery operated pump. We
18 typically put it on the belt of the person. Then we use a
19 flexible tube and then put a filter in what we call the
20 breathing zone of the worker, as close as we can to where they
21 breathe. Typically their lapel or collar.

22 Then we -- for asbestos, we take the cap off, which is
23 unusual. We don't do that for very many materials. And then
24 point it down so the dust doesn't fall on the filter; you're
25 collecting samples that are in the air. And then you

1 actually -- you physically at some point when you're finished,
2 you physically take the filter out of there and dissolve a
3 portion of the filter and actually count the fibers under a
4 microscope.

5 Back when we did the study in Bremerton, this is the
6 current methodology. The only difference -- the main -- there
7 are slight differences. The main difference in the method
8 that we used back then, we used a larger filter. Our
9 concentrations, our exposures were higher and a larger filter
10 helped disperse the fibers better. When the concentrations
11 started getting lower, we went to a smaller filter.

12 Q. Are there methods that tell you how to collect the sample
13 and how to analyze the sample?

14 A. Yes, absolutely there are. It's a slightly different
15 method now than it was then. We used 239. NIOSH is the one
16 that establishes sampling in analytical methods like that.
17 The current method is NIOSH 7400. Everyone must follow the
18 same method, use the same techniques. Otherwise, you can't
19 compare one sample -- a sample you collect and a sample
20 someone else collected.

21 Q. And those methods, are they specified also in the
22 regulations that actually set what the permissible exposure
23 limits are?

24 A. Yes, OSHA also has a method essentially. It's the same
25 as 7400 saying you must follow this method.

1 Q. Now, this was the methodology in 1975, 76, 77, 78, when
2 you were with the navy and then at Bremerton; is that correct?

3 A. Yes.

4 Q. Was there an earlier methodology for collecting and
5 analyzing air samples for asbestos purposes?

6 A. There was. Before this -- this light microscopy method
7 we used -- where we actually count fibers, we used a method
8 where you had a solution in a -- in what's called a midget
9 impinger. You put 10 milliliters of solution, typically like
10 distilled water, and then you would run air -- bubble air
11 through that filter -- or excuse me, through that solution and
12 you would analyze that solution. Actually count fibers and
13 particles in that solution.

14 Q. And how were the results reported? Were they reported in
15 fibers per CC, for example?

16 A. No, then they were reported as million particles per
17 cubic foot.

18 Q. And that's abbreviated MPP --

19 A. CF.

20 Q. -- CF.

21 A. That's correct.

22 Q. When was that methodology used for air samples for
23 asbestos operations versus the newer method that you described
24 to what you used in your Bremerton study?

25 A. I think the conversion was really in the late -- late

1 '60s and early '70s. I took one of the first classes if not
2 the first, I'm not sure, classes that NIOSH offered in
3 sampling and analyzing asbestos dust and that was using the
4 new method.

5 Q. When you did your -- back to the Bremerton study and the
6 operations that you sampled. Were there control measures in
7 place during the sampling for the different operations that
8 you studied?

9 A. There were some control measures in place for some of the
10 secondary manufacturing operations that we had put in place.
11 There were no control measures in place for any of the end
12 user activities.

13 Q. And so the control measures were on those operations on
14 the right that were done by just the one or two workers in the
15 shipyard.

16 A. That's correct, yeah. The only thing that could be
17 remotely considered a control in the end user activity was for
18 certain activities people, instead of throwing the debris on
19 the floor, they would put it in a plastic bag for disposal.

20 Q. Okay. And what was that referred to in the report?

21 A. That was one of the housekeeping techniques we used.

22 Q. Any other controls for the end user activities?

23 A. No.

24 Q. Would the housekeeping controls, putting the scrap in a
25 plastic bag, reduce the exposure a worker had for the

1 operation?

2 A. Putting it in a plastic bag wouldn't affect the exposure
3 whatsoever.

4 Q. So what did you learn about the exposure from working
5 with gaskets as an end user in the shipyard?

6 A. We learned that end users really didn't have exposure to
7 asbestos from gaskets. That they were well below the OSHA
8 standard. And for the most part, all of the samples were even
9 below the level at which medical monitoring would begin.

10 Q. And so we've projected a slide here that identifies the
11 different end user operations. I want to start here with the
12 process.

13 So in storage, it looks like you evaluated a couple of
14 different ways. And you collected 14 samples. Why would you
15 collect so many samples on just storage of gaskets?

16 A. Well, we wanted to -- we wanted our study to be
17 statistically significant. The navy asked that we conduct a
18 statistically significant sample, so we tried to get as many
19 as we could for different operations.

20 Storage, obviously, was something that was going on every
21 day so it wasn't difficult for us to get down there and
22 collect quite a few samples there.

23 Q. So looks like you collected a lot of samples on removal
24 as well.

25 A. Yes, we did.

1 Q. We talked about OSHA's permissible exposure limit. Did
2 OSHA also have a short-term exposure limit?

3 A. Yes, they did.

4 Q. During 1978 what was that limit?

5 A. That limit was ten fibers per CC.

6 Q. And we've indicated that on this graph; is that correct?

7 A. That's correct.

8 Q. And so these samples at the time of your study were well
9 below that excursion limit?

10 A. That's right. And that's the appropriate limit that you
11 would compare these standards to, these operations to because
12 they're short-term operations. They're not long-term
13 operations. You know, they're not -- it's not something
14 that's done on an 8-hour basis other than the storage, for
15 example.

16 Q. Did OSHA ultimately adopt a lower short-term exposure
17 limit?

18 A. They did.

19 Q. And what was that?

20 A. It's currently one fiber per CC.

21 Q. And it's indicated here since 1988. To your knowledge,
22 that hasn't been changed since 1988?

23 A. That's correct.

24 Q. Approximately 25 years. And so how do your samples
25 compare with the OSHA short-term exposure limits?

1 A. They're lower than even the current short-term exposure
2 limit.

3 Q. There's some photographs in your study; is that correct?

4 A. Yes, there are.

5 Q. I saw this photograph here, Figure 14. What was it in
6 relation to?

7 A. That shows a flange where the gasket has been removed,
8 but you can still see some of the gasket debris adhering to
9 the flange. So that's an example of a flange where they have
10 to do some clean up after the gasket has been removed.

11 Q. So the gasket has already been removed. They just have
12 to clean up that debris.

13 A. That's correct.

14 Q. Those type of activities, what did you find?

15 A. Again, we found very low levels of exposure no matter
16 which way they did those operations.

17 Q. Did you make recommendations in your report?

18 A. Yes, we did.

19 Q. I'd like to turn to those if we could. Is this a page
20 from that -- from the recommendation section?

21 A. Yes.

22 Q. Oh, I'm sorry. This is the clean up following removal
23 part, right. I want to ask you about this.

24 A. Yeah, that's not a recommendation. That summarizes the
25 results we got from that particular operation.

1 Q. All right. And after removal of gaskets from an object,
2 residual pieces of gasket material may have remained attached.
3 Figure 14, that's the photo in the bottom there, shows
4 residual material remaining on the flange.

5 And so it looks like hand scraping was the operation to
6 remove this residual material.

7 A. That's correct.

8 Q. And it indicates that less than 0.05 was the range and
9 the average for four samples. Can you tell us what does that
10 mean, less than 0.05?

11 A. Well, it -- you can't -- as scientists we never report
12 zero. You can never say that something is zero. But if we
13 had found any fibers for these samples, .05 is the lowest we
14 could have gone. So we don't know how much we had, but we
15 know it was less than that.

16 It's like trying to weigh a letter on your bathroom
17 scale. You can't say it weighs an ounce because your scale
18 doesn't go that low. You can say it's less than a pound
19 possibly. So that's the sort of thing we're doing here.

20 Q. So for cleaning up a flange face like Figure 14 using
21 hand scraping, were there any controls?

22 A. No.

23 Q. None of the housekeeping that you were talking about
24 before?

25 A. No.

1 Q. Now, let's look at the recommendations.

2 Hang on just one second.

3 THE COURT: Do you want to work on that and do it
4 tomorrow?

5 MR. HARRIS: We certainly could take a break if you
6 would like now, Your Honor.

7 THE COURT: All right. Why don't we go ahead and do
8 that.

9 MR. HARRIS: I'll be happy to. I'll be ready to go
10 in 45 seconds or 30 seconds.

11 THE COURT: Well, go ahead. We'll go for 15 more
12 minutes.

13 MR. HARRIS: Okay.

14 BY MR. HARRIS:

15 Q. Okay. We're back in business.

16 Recommendations. The first section, it's entitled "Work
17 practices and procedures for asbestos-containing gaskets;" is
18 that correct?

19 A. Yes.

20 Q. It says, "The recommended work practices and procedures
21 are based on reducing asbestos exposures to gasket workers to
22 below 0.1 fibers per CC."

23 Now, why did you choose that?

24 A. We chose that because that was the level at which medical
25 monitoring was to begin. We wanted to see if we could get

1 exposures below that.

2 Q. "The engineering controls for achieving this goal are
3 feasible and the necessity for medical surveillance for large
4 numbers of gasket workers is eliminated. If less stringent
5 goals, such as 0.5 and 2.0 fibers per CC are acceptable, the
6 work practices can be modified accordingly to summary table
7 XII. The control measures are as follows."

8 And did you make recommendations for each of the
9 operations that you studied?

10 A. Yes, we did.

11 Q. And so for secondary manufacturing, what those one or two
12 shipyard workers were doing, cutting gaskets or fabricating
13 gaskets all day, you actually recommended some controls; is
14 that correct?

15 A. We did. For secondary manufacturing we recommended local
16 exhaust ventilation and what we called a port vac which is a
17 high efficiency vacuum cleaner with a HEPA filter on it to
18 clean the areas.

19 Q. What is local exhaust ventilation?

20 A. It's a ventilation that draws the dust away from the
21 source where it's generated.

22 Q. All right. For the end user activities, did you make
23 other recommendations?

24 A. Yes.

25 Q. So for storage, any recommendations?

1 A. We didn't make any recommendations at all. We said it
2 would be nice if you put it in a plastic bag.

3 Q. For hand shaping?

4 A. The same thing. We said that the waste should go in a
5 plastic bag. And those were -- that wasn't to reduce
6 exposure. It was because the navy had disposal requirements
7 for asbestos-containing materials and so we wanted it already
8 put in the plastic bag to make those easier.

9 Q. Installation: No special controls are necessary.

10 And then for removal and flange clean up following
11 removal, what did you recommend?

12 A. The same recommendation as shaping. Just put the scrap
13 in a plastic bag for disposal.

14 Q. And does that reduce the exposures from those operations?

15 A. It does not.

16 Q. So Mr. Liukonen, I want to go back to the slide I jumped
17 up on a few minutes ago.

18 The committee's experts have lodged some criticisms of
19 this study. You're familiar with those?

20 A. I am.

21 Q. You read their reports that they filed in this case?

22 A. Yes, I have.

23 Q. And you've read their depositions?

24 A. Yes.

25 Q. Okay. I want to ask you about some of the things that

1 they've claimed.

2 First, we've heard that they've said that it's not a real
3 world study. Is it a real world study?

4 A. Absolutely. It was conducted in the shipyard under real
5 world work conditions.

6 Q. Was it simulated in any way? We've heard about Dr. -- or
7 we'll hear about Dr. Longo and how he's done some simulation
8 studies. Were you simulating any activities here?

9 A. There were no simulations whatsoever. Whatever happened
10 is what we tested.

11 Q. Is this a -- this wasn't a simulation study; it was a
12 field study.

13 A. That's correct. It's a field study.

14 Q. Now, are there limitations to field studies?

15 A. Absolutely.

16 Q. What are those limitations?

17 A. The main limitation from a field study is that you can't
18 control for other fibers from any other source so that you're
19 not a hundred percent sure that the fibers you're measuring
20 are from the gasket. They may be from an operation adjacent
21 or in the next space over.

22 Q. Another criticism is that no samples were taken aboard
23 ship. Is that true?

24 A. That is not true. Most of the end user activities were
25 taken aboard ship.

1 Q. Actually, does the report address that?

2 A. It does. It specifically says they were taken aboard
3 ship.

4 Q. This is Mr. Templin's report where he says, "A second
5 naval study," referring to your study, "concerning gasket
6 operations, this one conducted by the U.S. Navy and often
7 referred to as the Bremerton study, was reported in May 1978.
8 It was not, however, conducted in the field of workers under
9 actual conditions as contended by Boelter," et al. Do you
10 disagree with that?

11 A. Absolutely.

12 Q. Okay. On the samples aboard ship under project
13 definition, there's a line here. Can you tell us what they
14 say about where the samples were collected.

15 A. We said, "The majority of the samples were collected by
16 NRMC Bremerton at Puget Sound Naval Shipyard in various shops
17 and aboard ship."

18 Q. What about the end user activities, were those samples
19 collected aboard ship or in shops or...

20 A. Virtually all, if not all, of those were collected in --
21 aboard ship.

22 Q. "No major overhauls were going on at the shipyard." I
23 believe Mr. Beckett had said in his deposition maybe that all
24 the operations involving gaskets couldn't be sampled because
25 there were -- there wasn't a major overhaul going on in the

1 shipyard. Is that accurate?

2 A. That is not accurate. We had -- we had six dry docks.
3 They were almost always full. Our largest dry dock, dry dock
4 number 6, pretty much always had an aircraft carrier in there.
5 And as this document indicates, I looked it up to verify, and
6 indeed there was an aircraft carrier in the largest dry dock.

7 Q. In response to a FOIA request that Garlock's lawyers once
8 made, they produced a list of all the operations, all the
9 ships that came in through the Puget Sound shipyard over the
10 course of 30, 40 years. You've seen that document?

11 A. I have.

12 Q. You produced it, I believe, at your deposition. And
13 we've highlighted one of the ships that's listed there and the
14 dates that it was in service. It says, "CV-43 Coral Sea."

15 Is that an aircraft carrier?

16 A. It is.

17 Q. It came in for service on March 7, 1978, and left
18 February 7, 1979. How does that tell you that that's an
19 overhaul?

20 A. Because of the length of time that it was there. It was
21 there for nearly a year. So it was there for extensive work
22 and the timing is absolutely perfect for our study.

23 Q. When were you assigned to do the study and when did you
24 publish the study?

25 A. The original correspondence was in February of 1978. We

1 published it in May of 1978.

2 Q. And so this March 7, the first phase of overhaul is what?

3 A. It's absolutely perfect. That's when the work was being
4 done.

5 Q. "Controlled environment." I believe Mr. Templin also
6 said, "Rather, it was performed" -- referring to your study.

7 "It was performed within carefully demarcated barricades
8 placarded by large conspicuous asbestos dust hazard caution
9 signs, by employees wearing respirators and protective
10 clothing, and utilizing a variety of work practice,
11 engineering, and housekeeping controls."

12 Now, what's he talking about there?

13 A. Well, that's incorrect. There are some pictures. This
14 is an example. When we went to test, the person using the
15 sheer, he or his supervisor or someone had misinterpreted our
16 instructions for working with friable insulation. Decided
17 that those applied to gaskets, which they did not. But that's
18 an example of what he did. He put on protective clothing. He
19 put up a sign.

20 There you can see the rope and the sign. And had that
21 been friable thermal insulation we were working on, according
22 to our instructions you could work on this side of the sign
23 without any problem.

24 Q. Okay. So back in 1978 at a shipyard, when you were
25 talking about minimal controls to protect from potential

1 exposure to insulation, this was the rope and the sign that
2 you were talking about.

3 A. That's the rope and the sign, yes.

4 Q. So in 1978 there's not the tent or the plastic sheeting
5 that goes up to completely encase the area; is that correct?

6 A. That is correct. And when I arrive at a job site like
7 this, if someone wants to use more precautions than are
8 required, that's fine with me. I never tell them to use less
9 precautions.

10 Q. What operation is this?

11 A. This is machine sheering.

12 Q. Is this one of those secondary manufacturing operations
13 or is this an end user operation?

14 A. This is one of those secondary manufacturing operations.

15 Q. And this is one of the two people in the shipyard that
16 did that job?

17 A. This is kind of like two and a half. This job is done at
18 times so this is -- this is a little bit more than two. This
19 is a third person that does some of it.

20 Q. All right. Here is another picture out in the shipyard.
21 Looks like he may actually be working on Garlock gaskets
22 there; is that correct?

23 A. That's correct. That's the same individual and using the
24 same precautions. And this machine is called a nibler. I
25 think some people call it a fly cutter.

1 Q. Is this a secondary operation activity or an end user
2 activity?

3 A. It's a secondary manufacturing operation.

4 Q. All right. And so this is the work that was going on in
5 shops that was referenced at the beginning of the report?

6 A. That's correct.

7 Q. And so for the end user activities that were going on
8 aboard ship, you don't have photographs of people actually
9 working on ships; is that correct?

10 A. That's correct, none of our photographs were aboard ship,
11 I don't believe.

12 Q. Why would that be?

13 A. The shipyard was very tightly controlled on that sort of
14 issue and they didn't even allow their own photographers to go
15 take pictures aboard ship at this time.

16 Q. Okay. Now, Dr. Longo has made an interesting criticism.
17 The committee's expert, Mr. Beckett, evidently went down to
18 Dr. Longo's lab and had a conversation with him. And Dr.
19 Longo recants that in his report.

20 He says, "Bremerton's gasket study could not be used as
21 an indicator on all potential asbestos exposure removal
22 projects at Bremerton. Mr. Beckett said this was especially
23 true of the Bremerton study since it was done under abnormal
24 conditions because of the presence of a number of senior
25 personnel who were observing the study, a shipyard

1 photographer documenting the study, and housekeeping controls
2 utilized during the study."

3 And then he said in another deposition, "As Mr. Beckett
4 stated, this was a study that was -- where you had ship
5 photographers, you had all the brass watching the studies, and
6 they were not representative of what usually goes on with
7 gasket studies."

8 Now, you've read Mr. Beckett's deposition, correct?

9 A. I have.

10 Q. Do you recall what he said about that?

11 A. He said he never said that.

12 Q. Right.

13 A. Which is absolutely true. There were no navy brass
14 present during the sampling. There were industrial hygienists
15 or industrial hygiene technicians present during the sampling.
16 There were occasionally a shipyard -- or was occasionally a
17 shipyard photographer present to take some of these pictures,
18 but there were no navy brass present.

19 Q. And if navy brass were present or somebody was
20 influencing the results of your study, would that be something
21 that would be noted?

22 A. Absolutely. If we thought it wasn't representative or
23 real world work conditions, we would have noted it.

24 Q. And the purpose of -- one of the purposes of the study
25 was to determine whether people working with gaskets actually

1 needed to be medically monitored, correct?

2 A. That's correct.

3 Q. And if people were influencing the results of the study,
4 would you have to note that?

5 A. Absolutely.

6 Q. Mr. Liukonen, you worked in that shipyard for three
7 years; is that correct?

8 A. Four years.

9 Q. Four years. And you knew people that worked there.

10 A. Yes, I did. Bremerton is a small town.

11 Q. And did you know people that were actually working in the
12 shipyard that were working with gaskets?

13 A. Yes, I did.

14 Q. If you thought that they needed controls when they were
15 working with gaskets out in the field, would you have made
16 recommendations for controls?

17 A. Absolutely.

18 MR. HARRIS: Your Honor, would this be a good time
19 to take a break?

20 THE COURT: It's 5:30. Let's take a break and we'll
21 come back at 9:30 tomorrow morning.

22 (Evening recess at 5:29 p.m.)

23 *****

24

25

1 1 UNITED STATES DISTRICT COURT
2 2 WESTERN DISTRICT OF NORTH CAROLINA
3 3 CERTIFICATE OF REPORTER

4

5

6 6 I certify that the foregoing transcript is a true
7 7 and correct transcript from the record of proceedings in the
8 8 above-entitled matter.

9

10

 Dated this 24th day of July 2013.

11

12

 s/Cheryl A. Nuccio

13

 Cheryl A. Nuccio, RMR-CRR

14

 Official Court Reporter

15

16

17

18

19

20

21

22

23

24

25

26